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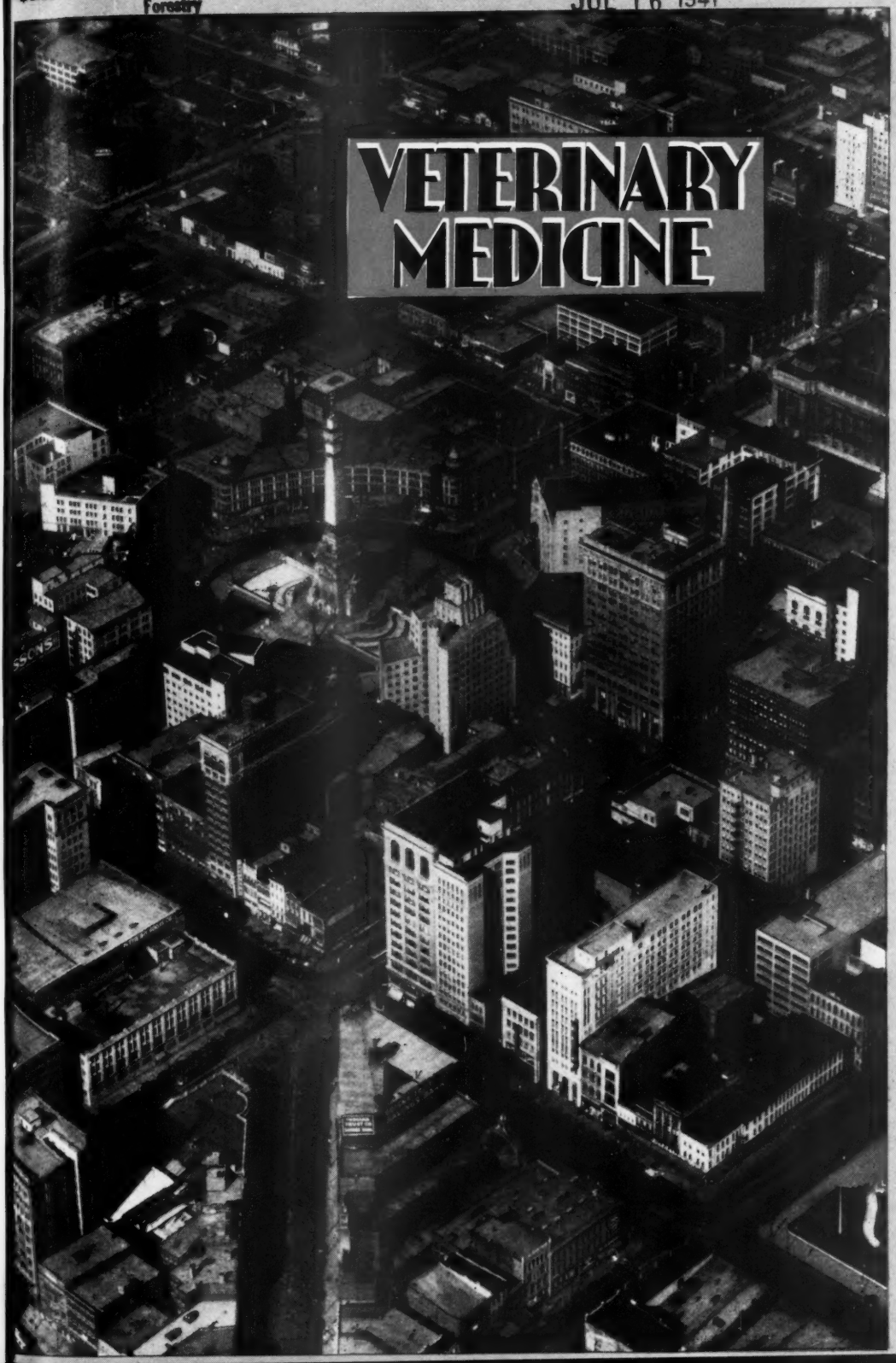

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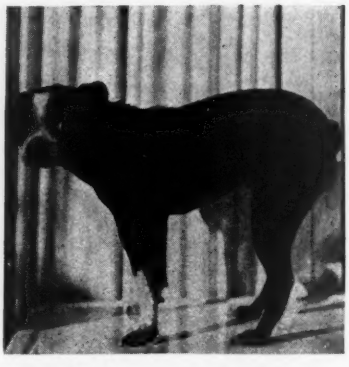

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CONTENTS

Veterinary Progress, Policies, and Comments 389-407

Cost of Eliminating Horses and Mules, 389; Maintaining or Losing an Interest in Live Stock, 389; The Composition of Milks, 390; Veterinary Staff, University of Pennsylvania, 391; A New Germ-killing Chemical, 392; B Vitamins Essential to Bacteria, 392; Eradicating Tuberculosis from Poultry Flocks, 392; The Newspapers Say, 393; Importance of Live Stock Production, 394; Poultry Statistics, 394; Live Stock in the United States, 394; Heat Resistant Cattle, 395; Do Good Scientific Books Become Obsolete?, 396; Attractions for Ladies at the Indianapolis Meeting of the A. V. M. A., 396; Best Features of Past Clinics to be Combined at the Indianapolis Meeting of the A. V. M. A., 397; The Practitioners' Association, 399; "Permanent Pastures Perpetuate Parasites," 403; Veterinarians of Indiana Hold 57th Annual Meeting, 404.

Lymphoblastoma in Dairy Cattle—L. S. Starr and Tyler Young 406

Equine Infectious Anemia in the United States—C. D. Stein 410

The Microscopic Detection of Bacterial Defects of Milk—C. S. Bryan 415

Vaccine Dosage in Canine Distemper—Leon F. Whitney 420

Recent Materia Medica XII. The Adrenal Hormones—Victor Lewitus 424

Phenothiazine Poisoning—C. D. Folse 430

Death of a Cow During an Intravenous Injection—S. W. Stiles 431

Injuries to Cattle Caused by Starlings—John W. McCoy 432

Tail Setting on the Way Out 433

Leucoplakia in a Dog—Robert B. McClelland 433

Canine Tetanus—C. C. Collins 434

Sulfapyridine in the Treatment of Feline Distemper—A. H. Bryan 434

Abstracts 435

Length of Gestation and Birth Rate in Cattle; Chemotherapy of Strangles; A Probable Agent for the Transmission of Fowl Paralysis.

Book Reviews 436

Animal Pathology; Eggs and Egg Products; Colorado's Poisonous and Injurious Plants.

THE FRONT COVER

As with the famed Indiana poet, James Whitcomb Riley, whose home was Indianapolis,

> "I AIN'T, ner don't p'tend to be, much posted on philosofy
> But thare is times, when all alone, I work out idees of my own
> And of these same there is a few I'd like to jest refer to you—"

one of them is—that members of the A.V.M.A. and their wives will have ample proof, throughout their stay in this city, that "Hoosier hospitality is more than a phrase." A plane's eye view of a part of the downtown section, including Soldiers and Sailors monument, is shown on our front cover.

VETERINARY MEDICINE

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Leading Publication in the Veterinary Field for More Than Thirty Years

VOL. XXXVI **AUGUST, 1941** **No. 8**

Tuberculosis is rarely seen in flocks less than six months old. If the young birds are allowed to mingle with old birds in the laying houses, a large number of the young birds may become infected and develop the disease later on.

Last year the state of Massachusetts paid the owners of live stock, including poultry, $26,000 for losses due to roving dogs. It is the opinion of the state Fish and Game Commission that dogs kill as many deer as do hunters—in excess of 3,000 annually. There are about 200,000 licensed dogs in Massachusetts and an even greater number of unlicensed.

Dr. Karl F. Meyer reported the discovery of psittacosis in both pigeons and chickens at a meeting of the American Epidemiological Society recently. The disease has been found in barnyards and pigeon roosts in California, Illinois and New Jersey. Doctor Meyer strongly suspects that many patients sick during the last several years with attacks of what has been diagnosed as "virus pneumonia" were victims of parrot fever.

In a study of liver abscesses in cattle undertaken at the Mayo Foundation, Rochester, Minn., over 10,000 such abscesses were examined. In this group the incidence of liver abscess was slightly higher in cattle fed sugar beet products than in other cattle. Although the *Actinomyces necrophorus* alone or associated with hemolytic streptococci is regularly found in these abscesses, there is doubt as to either organism's being the primary cause of the condition. Liver abscesses cause the condemnation of an enormous number of beef livers and are responsible for a heavy economic loss.

An error of judgment in diagnosis or treatment does not amount to malpractice unless it is so gross as to be inconsistent with the standard of due care.

There were five physicians among the signers of the Declaration of Independence: Oliver Wolcott (Connecticut), Lyman Hall (Georgia), Josiah Bartlett and Matthew Thornton (New Hampshire), and Benjamin Rush (Pennsylvania).

At the recent meeting in Chicago of the American Institute of Nutrition, the Mead, Johnson and Company prize of $1,000 for work on the vitamin B complex was awarded to Dr. Roger J. Williams, professor of chemistry at the University of Texas, for his "outstanding work on the isolation and identification of pantothenic acid."

The scientists in the Hall of Fame at New York University to date include: John James Audubon, Asa Gray, Louis Agassiz, Joseph Henry, Maria Mitchell, Matthew Fontaine Maury, and Simon Newcomb. The College of Electors, comprising 110 distinguished Americans, meets every five years to vote on new additions to the Hall of Fame. Sixty-five votes are necessary to election. The next election is in 1945.

In partial adrenal cortex insufficiency, where asthenia is the outstanding symptom, Davidson recommends that adrenal cortex extract be given to small animals either subcutaneously or intravenously at the rate of 0.5 to 2cc daily or on alternate days; in acute crises, doses of 2 to 4cc intravenously are recommended, in conjunction with the administration of fluids and sodium salts. Davidson regards overdosing as improbable.

Galen described a pestilence of unmistakable smallpox. Beginning in the Roman Army in A.D. 166, it spread over the whole of Italy and into Gaul, resulting in the death of more than half the population and almost all of the troops.—*U. S. Pub. Health Repts.*

* * * *

Food infection is as old as civilization itself. From his earliest creation down through the ages, man apparently acquired considerable knowledge concerning what was good to eat and what to let alone. Thirty-four centuries ago it was recognized that the bodies of animals that died a natural death were not fit for food, for Moses commanded that "Ye shall not eat anything that dieth of itself."—*T. G. Hull.*

* * * *

The dominance of magic over the life of early men cannot be overestimated. It played the part with him that religion, philosophy, and science occupy with us. It represented his first attempts to inquire into the secret workings of nature and to control and exploit them for his own benefit. Nowhere was the belief in magic stronger than in the case of disease, which was ascribed to mysterious and malignant forces. As magic was the precursor of science, it was the progenitor of medicine. The magician of the tribe was its medicine man. Quacks trade upon the subconscious remains of this faith in magic healing.

* * * *

When it was found that the blood of one human being could be injected into the circulation of another, the physician's armamentarium in the fight against disease was tremendously augmented. Today the indications for the application of transfusion are legion. Its use is no longer restricted to those persons who are desperately ill, but, as far as the pediatrician is concerned, transfusion is frequently utilized prophylactically to prevent the development of serious complications. There is no branch of medical practice in which transfusion is now not a common mode of therapy.—*J. A. M. A.*

One king of ancient Egypt had represented on his tombstone his favorite dogs: Gazelle, Greyhound, Black, and Fire Pot.—*Sci. News Letter*

* * * *

The origin of anthrax is lost in antiquity. About 1491 B.C. Moses is quoted as threatening Pharaoh with an epidemic among his live stock:

"Behold the hand of the Lord is upon thy cattle which is in the field, upon the horses, upon the asses, upon the camels, upon the oxen, and upon the sheep; there shall be a grievous murrain."—Exodus 9:3.

This "grievous murrain" is considered by many authors to have been anthrax. The severity of the outbreak is indicated by the statement that all the cattle of Egypt died. —*T. G. Hull.*

* * * *

Until late in the nineteenth century, veterinarians, animal husbandmen, and governments were primarily concerned with the great animal plagues: rinderpest, foot-and-mouth disease, contagious pleuro-pneumonia, and anthrax of cattle, which swept relentlessly over state and nation, causing appalling losses and imperiling the food supplies of the inhabitants. Finally these have been or are being brought under control. Meanwhile grave threats to physiological reproduction were being insidiously evolved.—*W. L. Williams*

* * * *

Lesions found in the bones of Egyptian mummies have shown that tuberculosis was very prevalent in the time of the Rameses, thirteen centuries before Christ. The Hindus in India, in their early writings contained in the Veda in the fifteenth century B.C., and the Parsees at about the same time, also made reference to the disease. From that time down through the years, wherever the white man has left his record, accounts of the ravages of tuberculosis may be found under one name or another. Tuberculosis in animals was likewise recognized in very early times. The Mosaic Law prohibited the use of the flesh from animals suffering from "wen or scurvy." The Talmud of the first century A.D., the Mishna of the third century, and the Gemara of the fifth century, also contain similar prohibitions.—*T. G. Hull.*

Cost of Eliminating Horses and Mules*

The importance of horses and mules to our nation may be still better understood if we think of what would happen if they were all wiped out of existence. Mechanized equipment costing over 8 billion dollars additional would be required to take the place of our present horses and mules, which were valued on January 1, 1941, at $1,155,000,000, and would not do our work as well as the animals; in fact, could not do all that horses and mules do.

Eight billion dollars is almost 3 billion dollars more than the combined value of all horses, cattle, sheep and hogs on January 1, 1941; it is about 7 times our present investment in all horses and mules.

Replacement costs, annually, would exceed a billion dollars a year, even on a basis of 8 years' life for mechanized equipment, which is too liberal. Replacements needed to maintain present numbers of horses and mules are about 1 million animals per year, costing not over $80 per head, when 3 years old and ready to work.

To substitute mechanized equipment for the approximately 15,000,000 horses and mules we now have, is a proposal to replace our present farm power equipment with new equipment costing seven times as much in original investment and more than 10 times as much for yearly replacements—a billion dollars a year against about $80,000,000 a year for animal replacements.

And this is not all. Our farm surplus problem would be increased. Sixty million acres of fertile cornbelt land or more—its equivalent in lands of lesser fertility—would be diverted from present use in producing and maintaining horses and mules, to the production of farm products which would have to be sold for cash, thereby depressing still further markets already overloaded with foodstuffs in grains, dairy products and meat animals. What could be grown on 60 million acres additional that would find a ready market and show a profit to farmers?

At the present time, when factories and all skilled mechanics are urgently needed to produce tanks, airplanes, guns, munitions and other things needed for our national defense a proposal to eliminate all horses and mules is so absurd, so lacking in timeliness, as to be ridiculous.—*Wayne Dinsmore.*

* The Ford Motor Company has suggested recently, in widely distributed advertising in farm papers, that horses be completely eliminated.

* * * *

Maintaining or Losing an Interest in Live Stock

The man who operates his farm with horse power primarily, maintains the interest that he already has in livestock. It is something that we can't put on paper—something that a man feels — something that a man knows but can't explain. The man who turns to tractor operations in so many, many cases on these small farms finds it necessary to give up all or practically all of his horses, and I have seen many of these farmers who a few years before I had classed as good livestock men, gradually lose interest in their livestock. I have seen them become more and more cash grain farmers, piece workers if you please, and I have seen their income and their security on the land decrease with that change in attitude and interest. Show me a farmer with enough horses, that are good horses, horses that he is proud of, horses that he sees win glances of envy and admiration from his neighbors when he goes threshing, and you will show me a farmer that takes good care of his other livestock and makes a profit at it. You will have shown me a farmer that is going to be able to pay his bills and pay his loan and be secure on the farm in the years to come. —*Donald Hammerly, Iowa State Supervisor U. S. Farm Security Administration.*

"Combining" Wheat in the Northwest

The Composition of Milks*

TABLE I. AVERAGE COMPOSITION OF MILKS OF CERTAIN MAMMALS

Species	Water	Protein	Fat	Lactose	Ash
Woman	87.43%	1.63%	3.75%	6.98%	0.21%
Cow	86.21	3.77	4.45	4.86	0.72
Goat	87.14	3.71	4.09	4.20	0.78
Ewe	82.90	5.44	6.24	4.29	0.85
Egyptian buffalo	82.09	4.16	7.96	4.86	0.78
Chinese buffalo	76.80	6.04	12.60	3.70	0.86
Philippine carabao	78.46	5.88	10.35	4.32	0.84
Camel	87.61	2.98	5.38	3.26	0.70
Mare	89.04	2.69	1.59	6.14	0.51
Ass	89.70	2.10	1.50	6.40	0.30
Reindeer	63.30	10.30	22.46	2.50	1.44

TABLE II. DISTRIBUTION OF NITROGEN IN HUMAN AND IN COW'S MILK

Kind of Milk	Total Nitrogen	Percentage of total nitrogen		
		Casein nitrogen	Noncasein protein nitrogen	Nonprotein nitrogen
Human	0.1749%	34.3%	30.0%	35.2%
Cow's	0.497	76.1	18.0	5.9

TABLE III. AVERAGE COMPOSITION OF MILK OF FIVE BREEDS OF COWS

Breed	Water	Total Solids	Fat	Protein	Lactose	Ash
Guernsey	85.13%	14.87%	5.19%	4.02%	4.91%	0.74%
Jersey	85.31	14.69	5.18	3.86	4.94	0.70
Ayrshire	86.89	13.11	4.14	3.58	4.69	0.68
Holstein	87.50	12.50	3.55	3.42	4.86	0.68
Shorthorn	87.43	12.57	3.63	3.32	4.89	0.73

TABLE IV. AVERAGE YEARLY PRODUCTION OF MILK AND BUTTERFAT OF DIFFERENT BREEDS

Breed	Advanced register or register of merit				Herd-improvement test			
	Cows and heifers	Milk	Butterfat		Cows and heifers	Milk	Butterfat	
			Quantity	Test			Quantity	Test
	Number	Pounds	Pounds	%	Number	Pounds	Pounds	%
Ayrshire	9,022	10,446	416	4.0	20,571	8,351	338	4.0
Brown Swiss	939	13,643	548	4.0	984	8,577	352	4.1
Guernsey	49,212	10,095	502	5.0	5,229	8,629	425	4.9
Holstein-Friesian	56,007	16,005	546	3.4	52,369	11,101	380	3.4
Jersey	56,789	8,556	459	5.36	27,364	6,797	359	5.3

TABLE V. RELATION BETWEEN THE PROTEIN, FAT, AND ENERGY VALUES OF MILK

Fat (per cent)	Overman, Sanmann, and Wright			Haecker			Ohio analyses,
	Protein		Energy	Protein		Energy	
	Found	Calculated	calculated	Found	Calculated	calculated	protein calculated
	%	%	Calories per lb.	%	%	Calories per lb.	%
3.0	3.07	3.20	290	2.68	2.66	278	2.87
3.5	3.39	3.40	315	2.81	2.86	306	3.08
4.0	3.54	3.60	341	3.08	3.06	336	3.29
4.5	3.83	3.80	367	3.27	3.26	365	3.50
5.0	4.02	4.00	392	3.45	3.46	390	3.71
5.5	4.18	4.20	418	3.65	3.66	415	3.92
6.0	4.50	4.40	443	3.82	3.86	440	4.13
6.5	4.63[1]	4.60	469	4.02	4.06	467	4.34
7.0	4.91	4.80	494	4.22	4.26	492	4.55

* *U. S. Department of Agriculture Yearbook*, 1939.

(Continued at bottom of next page)

FACULTY OF THE SCHOOL OF VETERINARY MEDICINE, UNIVERSITY OF PENNSYLVANIA

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The following members of the Faculty were absent at the time the photograph was taken: B. Lucke, M.D., Dr. P.H., Professor of Pathology; M. McCutcheon, A.B., M.D., Professor of Pathology; H. L. Ratcliffe, M.S., Sc.D., Assistant Professor of Parasitology; W. E. Ehrich, M.D., Associate in Pathology; G. P. Williams, Jr., A.B., L.L.B, Instructor in Veterinary Jurisprudence; D. G. Lee, V.M.D., Instructor in Anatomy; D W. Crisman, V.M.D., Instructor in Veterinary Hygiene; W. E. Evans, B.S., Assistant Instructor in Physiological Chemistry; M. J. Deubler, V.M.D., Assistant in Veterinary Pathology.

Composition of Milks

(Continued from preceding page)

TABLE VI. THE COMPOSITION OF THE ASH OF MILK[1]

Mineral	Ranges of values found by several European investigators	Values reported by Babcock[2]	Mineral	Ranges of values found by several European investigators	Values reported by Babcock[2]
	%	%		%	%
Potassium	14.6 to 23.9	20.8	Iron	0.035 to 0.280	0.001
Calcium	14.2 to 20.5	14.3	Phosphorus	9.44 to 12.8	10.6
Sodium	1.93 to 8.2	7.4	Chlorine	12.2 to 16.4	14.3
Magnesium	0.72 to 3.01	1.46	Sulphur	1.53 to 2.46	2.30

[1] Data given here are calculated from data in *Fundamentals of Dairy Science*, by Associates of L. A. Rogers (1935).
[2] In *Food Inspection and Analysis, for the Use of Public Analysts, Health Officers, Sanitary Chemists, and Food Economists*, by Albert E. Leach (1920).

Only in theory can ordinary repair processes be augmented by creating further injury. It may be possible to excuse the cautery on the ground of expediency, but it is unlikely that scientific reasons can ever be adduced to justify such violent and paingiving treatment.—*B. G. Catmur,* in *Vet. Rec.*

A New Germ-Killing Chemical

A chemical from the earth itself, the most powerful germ killer yet discovered, has cured in a remarkably short time an army of difficult and dangerous human ills, according to reports from the Mayo Clinic and the Massachusetts Memorial Hospitals to the American Society for Clinical Investigation meeting in Atlantic City, New Jersey.

The new chemical is gramicidin. It is produced by bacteria that live in the soil. Its discovery was announced in 1939 by Dr. René J. Dubos, of the Rockefeller Institute for Medical Research, but the reports presented at the meeting are the first on the use of gramicidin in human patients.

Empyema from pneumonia, sinusitis, bladder infections and dangerous and hard-to-cure staphylococcus infections in wounds are the conditions that this new chemical remedy cured in remarkably short time. Infected wounds were freed of all bacteria within twenty-four hours after gramicidin treatment, following which the wounds rapidly healed, was reported by Dr. Charles H. Rammelkamp and Chester S. Keefer, of Boston. "This is a distinct advance inasmuch as former antiseptics would not rid the wound of bacteria without seriously damaging the tissues."

Sinus infections were cleared up within 48 hours, according to Dr. Wallace E. Herrell and Dr. Dorothy Heilman, of the Mayo Clinic. Severe bladder infections which the sulfa drugs do not affect were cured within one week. Gramicidin is the most highly effective agent yet discovered for stopping the growth of bacteria. Trials on twelve patients at the Mayo Clinic were reported in detail, although three more have also benefited from the new chemical.—*Science.*

B Vitamins Essential to Bacteria

Investigations with microorganisms have made it seem apparent that the "B vitamins" (or at least some of them) are more fundamental and of more far-reaching importance than the other vitamins, in that the evidence indicates that they may be present and function in all forms of life from the highest to the lowest. Microorganisms have played a leading role in the discovery of five of the eight members of the vitamin B complex which have been isolated or identified as possessing vitamin properties, *i.e.*, pantothenic acid, nicotinic acid, biotin, inositol, and *p* aminobenzoic acid. Though each individual substance presents its peculiar problem it is safe to say that microbiological methods are destined to play a highly important part in the future determination of "B vitamins." The microbiological methods can be rapid and fairly accurate, and at the same time they can be applied to extremely small amounts of material.—*Prof. Roger J. Williams.*

Eradicating Tuberculosis from Poultry Flocks

Experiments which have been conducted jointly by Iowa farmers and the Iowa department of agriculture show that avian tuberculosis can be eradicated from farm flocks by:

1. Killing and burning all birds that show symptoms of the disease.

2. Hatching and brooding all young chicks artificially so they will not come in contact with old birds that may be carriers of the disease.

3. Using clean ground for brooding and rearing chicks.

4. Selling all old birds early in the summer and fall as they slack up in production so they will not be on hand to mingle with the young birds and give them the disease.

5. Cleaning and disinfecting all hen houses thoroughly before young birds are put into them.

6. Cultivating yards wherever possible, rotating them so as to have fresh ground that is not contaminated with disease germs, worm eggs, etc.

7. Removing all hiding places where sick birds may crawl to die.

The Newspapers Say—

Calfhood vaccination has been recognized officially as an adjunct or supplement to the test-and-slaughter plan for the control of this disease (brucellosis in cattle) but not as a substitute for it, according to Dr. C. R. Donham, head of the Veterinary Department of Purdue University. "Let us not make the mistake of too hastily discarding tried and proved methods for the control of animal disease," the veterinarian said.—*Goshen* (Ind.) *Democrat*

Dairy farmers at the first session of the farmers' evening school were frankly advised to resort to calfhood vaccination in their efforts to control Bang's Disease by Dr. John L. Cox, federal veterinarian in charge of testing in this area, who states: "The test-and-slaughter method has proved too difficult and expensive."—Harvard (Ill.) *Herald*

Orange Grove, Tex.—In 1906, when the Federal, state and local authorities began tick eradication work, 985 counties in 12 southern states, Missouri, Tennessee, and Southern California were under quarantine because so many of their cattle were infested with ticks. Today, after continued dipping and other control measures, there are only 12 counties under tick quarantine—eight in Texas, at the Mexican border, and four in Florida, where ticks persist on wild deer that avoid the dipping vats.

Des Moines, Ia.—Poultry paralysis causes an estimated national loss of 50 million dollars a year to poultrymen, according to Dr. C. A. Brandly of East Lansing, Mich., Senior Pathologist at the U. S. Regional Poultry Laboratory.

Some portions of the daily press in Britain print heated controversies over the right of the dog to live in wartime. "Down with all dogs! They guzzle while our hens starve! Dogs in wartime must die!" scream headlines, only to be followed in endless succession by tales of the courage and fidelity of dogs during air raids and of timely warnings that have saved lives; of rescue work which dogs have assisted and sometimes carried out unaided. Dogs are news in bomb-strafed England.

Lansing, Ia.—Pullorum disease control under the National Poultry Improvement Plan is progressing rapidly as the season of incubation and hatching approaches, says Dr. A. L. Howard, Assistant State Veterinarian in charge of poultry disease inspection.

According to Howard, hatching capacity of cooperating hatcheries has nearly doubled over that of the previous year with 88 hatcheries and independent breeding flocks now participating in the plan in Iowa. Howard reports with the pullorum testing program now in full swing, 275,000 breeding hens in over 1,200 flocks have been tested so far this season, and intimates that approximately one-fourth of the chicks to be hatched in Iowa this coming season will have come from pullorum tested flocks.

Mediapolis, Ia.—Dr. George Senior emphasizes that poultry disease can be found in almost every flock in the state. Diseases cause a 35% death loss to poultrymen each year, and in addition, result in lower quality products. . . . In fact, 15% of all birds sold are discarded as being unfit for human consumption. Much of the latter loss is due to tuberculosis and tumors. . . . Tuberculosis is present in 69% of the Iowa farm flocks and 16.8% of all old hens reacted to the tuberculin test.

Pomona, Calif.—Agricultural Expert C. E. Howell, Manager of the W. K. Kellogg Arabian horse ranch, heralded phenothiazine as a likely medium to rid horses and all other domestic animals of their internal parasites.

"Experiments on our horses," he said, "indicate that it is efficient, easily administered, nontoxic and cheap—which means that for the first time the average farmer may have a method which is not too expensive to rid his horse of parasites that weaken them."

Phenothiazine is a light green powder which turns darker upon exposure to air. It was mixed in with feed and administered to 45 purebred Arabian horses, including weanlings, yearlings, stallions, geldings, mares with foals, and mares pregnant six to ten months.

"The treatment was 100% effective against strongyles—small stomach worms—the most common type of infestation," said Howell. "It was partially effective against pin worms, but apparently had no effect upon bots.

"One of the treatment's biggest advantages is its safety," he declared. "Another is the inexpensiveness of the treatment. One pound, enough for eight horses, costs $1.50."

The paper goes on to explain that Howell gave phenothiazine its first trial in horses last year, and that now the N. Y. State Veterinary College is trying it.

Importance of Live Stock Production

No single phase of the national defense program is of greater present day significance than the problem of insuring an adequate diet for America's 131 million men, women, and children. A survey recently conducted under the direction of national defense authorities revealed that a third or more of our population is living below the safety line of what is considered an adequate diet. This means that the diet of this large group is deficient in the factors necessary for building and maintaining health.

The importance of meat in helping to solve this national nutrition problem cannot be overestimated. Research studies in recent years have established the high rank of this food product as a source of nutritive and health elements. On the basis of average servings, meat is a leader among foods, as a source of protein, iron, phosphorus, and calories. Within the past year or two, it has been discovered that meat is the richest known food source of the important B group of vitamins—the best known of which are thiamin, riboflavin and nicotinic acid. These are the vitamins which are at present in the spotlight of national advertising, and are being stressed by national defense experts.

Poultry Statistics

The production of poultry and eggs in the United States has hardly kept pace with the increase in population during the last decade, according to the figures of the sixteenth decennial census. While the number of persons living in this country increased about 7% during the period 1930-1940, the only major item of poultry production to show an increase greater than this was turkeys, the number raised during 1939 being 27,933,756, as compared with 16,794,485 during 1929. The production of chickens and chicken eggs was somewhat lower at the end of the decade, the comparative figures for chickens raised being 660 565 663 for 1939 and 673 092 052 for 1929, and for eggs 2 391 091 510 dozens and 2 689 719 158 dozens, respectively.

While most of the country showed a decline in the production of both chickens and eggs, the Atlantic seabord states almost all showed an increase, the New England states leading the others with an approximately 50% increase in both chicken and egg production, according to the census.

Live Stock in the United States

The sixteenth decennial census shows a marked decrease in numbers of horses and mules on farms and ranches, a slight decrease in the number of sheep, but increases in cattle and hogs, as compared with ten years ago.

The decade has witnessed a further adjustment of farm power to the use of automobiles, trucks and tractors on farms in the United States. A total of 13,931,531 horses, mules and colts were enumerated as of April 1, 1940, compared with 18,737,524 as of the same date in 1930. The number of colts in some areas, however, indicates a slight upward tendency.

Cattle and calves enumerated April 1, 1940, were 60,674,734 head compared with 54,250,300 ten years ago. The increases were quite general over the United States, although those areas where cattle numbers were worst affected by the droughts since 1934 have not yet been restocked to normal numbers. This census showed 21,936,556 cows milked on United States farms in 1939 compared with 21,124,221 in 1929.

The 1940 census found a total of 34,037,253 hogs, of which 7,987,641 were reported to be sows or gilts which farrowed or were to farrow for the 1940 spring pig crop. This compares with 32,793,628 hogs enumerated as of April 1, 1930, of which 7,673,741 were reported as sows and gilts bred for spring farrowing. There has apparently been a quite marked increase in the number of swine in the Southern states; some of the western Corn-Belt states, where the number of hogs has been depleted by droughts, have not recuperated to normal.

Sheep and lambs over six months old totaled 40,129,261, compared with 41,780,146 in the 1930 census. The mountain and Pacific coast states showed the greatest reduction in sheep numbers; the west south central states (Arkansas, Louisiana, Oklahoma and Texas) states showed a marked increase since 1930.

Heat Resistant Cattle

We have long been familiar with heat resistant bacteria and even with thermophiles. It has been recognized that races of men possess different degrees of tolerance for tropical heat since history began to be recorded. Of course everyone in the Deep South knows that mules can stand the heat of their long summers better than horses. That this is because the mule eliminates less salt in his perspiration than does the horse and thus does not so soon lower the sodium threshold in his blood, and that it is the sodium deficiency that causes the symptoms of overheat, are facts not so well known, but equally well established.

Until recently but little thought has been given to the effects of high atmospheric temperatures on cattle although they are equipped with a much less efficient heat regulating equipment than are the solipeds or man, and suffer obvious discomfort on the hottest days even in the northern of the Great Plains states. It has long been a matter of common observation in the section of long, hot summers that purebred animals of the beef breeds, under the best conditions, make about only 80% of the gain made by similar animals in the northern states. This has been attributed to poor care, annoyance of insects, less nutritious feed, or a combination of these factors.

About 40 years ago because purebred shorthorns and Herefords did not do well on the King Ranch in the extreme south of Texas, the Klebergs imported some Brahma cattle to cross upon them. The result was not very satisfactory. To begin with, the Brahma is an inferior beef animal. Further, it is wild and unmanageable. The first and second crosses, although better beef strains, retained the undesirable characteristics of wildness and unmanageability. However, they thrived on the King Ranch and the development of a new breed was undertaken. Now, after 30 years, the Klebergs have developed a highly satisfactory breed for that section. It is called the Santa Gertrudis for the division of the ranch on which it was developed. Klebergs are using 4,000 bulls of this breed on the ranch at the present time.

The King Ranch comprises 1400 square miles, nearly 900,000 acres. It is larger than Rhode Island and two-thirds the size of Delaware. It was formerly much larger than at present.

Cowhands, mechanics and laborers to the number of 450, plus their families, live on the ranch. Eighteen hundred cow horses are required in its operation. There are 1500 miles of fence on the ranch.

The cattle on the ranch number 65,000, brood mares about 500. Breeding operations include the development of Santa Gertrudis cattle, a strain of all purpose saddle horses, and a strain of Thoroughbreds.

Some two years ago scientists at the government research station at Jeanerette, Louisiana, undertook some experiments to ascertain the effect of the high temperatures and brilliant sunlight of that section on cattle. Purebred beef cattle from the north were pastured and fed under the same conditions that obtained for crosses between cows of the same breed and Brahma bulls. On a clear day when the atmospheric temperature was 96°F. temperatures among the northern cattle reached 107.5°F. and respiration in resting animals 140 per minute. Under the same conditions Brahmas and Brahma first crosses showed no elevation of temperature above normal and only slightly increased respiration. On hot days cattle of the common beef breeds grazed but little and then sought the shade. Brahma crosses grazed a normal amount and often lay down in the sunshine to ruminate, seemingly entirely unaffected by the heat. The northern cattle made only about 75% of the gain normal for northern sections of the country. Animals of the Santa Gertrudis breed made as rapid gain as is to be expected of the common beef breeds anywhere.

This development of a breed of cattle unaffected by the long, hot summers of the Deep South and one that will produce gains equal to that of the best breeds in the most favored localities promises to be of importance to the live stock industry of the South second only to eradication of the fever tick.

Goat milk production in Massachusetts alone is now a $2,000,000 enterprise. The retail price varies from 40c to 75c per quart. —*Amer. Fur. Breeder.*

* * * *

The use of a new drug (Urogastrone) for stomach ulcers was reported recently to the American Gastroenterological Association, by Dr. David J. Sandweiss of Detroit. Urogastrone was administered to 125 dogs suffering from stomach ulcers and all were relieved.

* * * *

Do Good Scientific Books Become Obsolete?

A scientific fact or theory set down in a bound book will always have value as the contemporary conception of the subject. Needless to remind the faithful scientist that the essence of original research is often a valuable guide in future investigation regardless even of faulty conclusions. The mistakes of the past set down in books today and called obsolete tomorrow are paths around the pitfalls.

The advance of science is a step-by-step process, seldom a single leap to the top rung. The research worker looks to books for the noteworthy teachings of his honored predecessors, perhaps more than in the daily output of the monographs characterizing present day medical literature. The idea that books rapidly become obsolete is, therefore, but a near truth. Portions of them are indelible and all of their text is helpful to the watchful scientist bent upon keeping up with current developments. They supply the background upon which current developments are founded.

In veterinary medicine, it would not be gainsaid that periodical magazines are a necessity even to the bookworm. Yet, to dispense with books where all of this knowledge is registered for permanent record would be a long step backward. The numberless monographs, bulletins and brochures published for the various branches of medicine today, even though carefully indexed and filed (and they seldom are) can never replace the convenience and the dignity of a medical library in the office or home.

The answer to our headline is, therefore, decidedly, No.

Attractions for Ladies at Indianapolis Meeting of A.V.M.A.

A tour through colorful South America, via illustrated technicolor moving pictures, will be the highlight of the luncheon program of the Ladies Auxiliary of the American Veterinary Medical Association, during convention week.

Julia Bock Harwood, widely-known traveller and lecturer, will speak on "South America—A Land of Contrasts." Illustrating her talk with technicolor movies and presenting models dressed in authentic native costumes, Mrs. Harwood will take her audience with her from New York City to Rio de Janiero, "most beautiful port of the Seven Seas," to Brazil, to the great coffee port of Santos, to Montevideo, Buenos Aires, Chile, Peru, Ecuador and Columbia.

Murat Temple and Shrine Theatre, Indianapolis, Ind., where all sessions of the 78th Annual Meeting and the Clinic of the AVMA will be held Aug. 11-15, 1941. The commercial and educational exhibits also will be displayed in this building

A card party and general session of the Auxiliary will follow Mrs. Harwood's talk.

Many other social events are being planned to make the 1941 convention of the A.V.M.A. a banner get-together for the ladies as well as for their husbands. The Banquet and Ball, the President's Reception, automobile races, a visit to the Real Silk Hosiery Mills, the world's largest hosiery factory, and numerous sightseeing trips are on the entertainment calendar.

Best Features of Past Clinics To Be Combined at Indianapolis Meeting of A. V. M. A.

A combination of the finest features of clinics at all past conventions of the American Veterinary Medical Association will be offered to veterinarians attending the 78th annual meeting of the A. V. M. A. in Indianapolis, August 11 through 15.

According to the tentative program, clinics will be held on Friday of convention week, in the morning and afternoon. Both large and small animal clinics will be held in the spacious convention halls of the Murat Theatre and Shrine Temple, scene of all business sessions of the convention.

The Murat Temple offers unusually adequate facilities for making the clinics the most interesting and instructive ever witnessed by A. V. M. A. members. Every veterinarian will have a clear view from all parts of the large rooms and will be able to watch in comfort the details of the clinical demonstrations.

Because of the importance of conserving the nation's food supply in this time of world crisis, the sheep and swine clinic will receive more than its usual share of attention. Keen interest will center around the many types of enteritis demonstrated, the various modes of swine anesthesia, parasitisms, cesarean section in the sow and the demonstration of pig anemia.

Popular demonstrations such as artificial insemination, heifer spaying, mastitis testing and teat and udder surgery will be shown in the cattle clinic.

Clinical demonstrations on horses will include the more common conditions that confront the practitioner in his daily work. These demonstrations include nerve blocking for both head and limb surgery, eye disease demonstrations, tracheal disturbances, pregnancy tests, and diagnosis and correction of skin ailments.

The poultry clinic will comprise an extremely wide range of disease conditions. There will be demonstration tests for diagnosis of specific diseases such as tuberculosis, pullorum disease, and fowl cholera carriers, and a practical and unique bleeding machine will be shown in operation. Poultry anesthesia will be demonstrated.

For those interested in small animal practice, the clinic will be attractive, ranging from plastic surgery to transfusions. An extremely variegated program covering all of the common ailments of pet animals will be presented.

A striking airplane view of the convention city appears on the front cover.

A Charge for Seals of Approval

Officials of the A. V. M. A. are emphatic in their protests that no charge is made for its seals of approval for dog food. It is true the A. V. M. A. as an organization receives no income from this source, but the fact remains that it costs the dog food manufacturers nearly $1,000 a year to have such seals. They must contract to pay this amount if use of the seal is permitted. No statement of distribution of this money has ever been published. An "allotment" for the funds has been reported to the association, which is sufficiently commented upon by saying it is an insult to the intelligence of the members.

The unwisdom of an inspection paid for by the inspected is so well recognized that such a proposal is seldom made in good faith. However, because of inexperience, this plan is sometimes tried with the best of motives. A case in point is the recent experience of the American Humane Association in its early attempts to prevent cruelty to animals in the filming of motion pictures, particularly in the Hollywood studios. To quote its official organ, *The National Humane Review:*

"Some pictures which were made in the old days were examples of unmitigated brutality. Horses were killed with disregard of law and public opinion. Wild animals were forced to fight each other. Domestic creatures were subjected to any devices to gain effects. Studio vied with studio, director with director, to produce the biggest thrill, to make the audience gasp.

"There occurred numerous outbursts that reached national proportions. The thrill aspect threatened to become a liability.

"The industry, through the Hays office, made an attempt to cope with the situation. No matter what others may think I regard that attempt as having been made in all sincerity. The Hays office requested the producing companies to have a humane officer present whenever animals were being used, and to obtain from the officer a certificate attesting the fact that no cruelty took place in the filming. As this was to be for the protection of the studio the officer would be paid at the rate of ten dollars per day.

"The plan failed from the outset. It failed *because the studios were paying the bill.* In California $150 a month is considered fair remuneration for humane officers and keen competition developed for duty that yielded twice that rate of pay. *Small groups even formed themselves into humane societies for the prime purpose of capturing business.* In Los Angeles County alone there are twenty-four chartered humane societies and five humane commissions. [Italics ours.]

"Thus a system that was conceived in good faith was doomed. You have only to mingle with a group of five or six humane officers to know why. And the explanation is not to our credit."

Briefly, the scenes in which allegedly cruelty was inflicted continued as before, except that now the producers had the statement of an accredited humane agent certifying that no cruelty was inflicted.

In time, the Hearst chain of newspapers ran a series of editorials and articles on cruelty to animals in the filming of motion pictures, and both the motion picture industry and the American Humane Association had a first class scandal of national proportions on their hands.

Again quoting from *The National Humane Review:*

"The substantial anticruelty societies in California were chagrined by the pseudo humane societies that sprang up in the southern part of the state, ostensibly to deal with cruelty cases within a given area, but primarily to provide humane officers who could collect $10.00 a day from studios making pictures with an animal sequence. The results are graphically set forth in your editorial [William Randolph Hearst's]. The humane movement as a whole was placed under suspicion and some members of the film industry stultified themselves by taking advantage of the situation and produced scenes that led to animals being badly hurt or killed.' '

After a searching investigation by two officials from the national headquarters of the humane association an entirely new arrangement was decided upon. One regulation in the new arrangement which the A. V. M. A. House of Representatives should ponder is (again quoting from *N. H. R.):*

"The industry was advised that thereafter no certificate of a humane officer to the effect that a picture was made without cruelty to animals would be acceptable to the Association unless he had been designated by it and his wage *paid by the Association.* . . . Under no circumstances must the film industry be allowed to pay the agent for his services."

The new program also provides that no firm that films animal pictures may make a contribution to the national nor to any local humane organization. Not all producers have as yet accepted the new arrangement, but progress is being made and there is prospect that damaged reputations may in time be retrieved.

Specifically, the humane associations, well intrenched as they are in public favor, could not take the film producers' money and certify to their humaneness, without engendering a public scandal. What prospect then is there that the American Animal Hospital Association can take the dog food manufacturers' money in far greater amount and issue them seals of approval without giving rise to similar public disapproval? If this happens the A. V. M. A. will be the principal victim.

It is the opinion of the writer that the A. V. M. A. dog food seal of approval already provides amply for a newspaper scandal if a resourceful and imaginative reporter sets out to construct one, and that this material is being added to as the high-pressure sales campaign to bring additional dog food manufacturers into the scheme progresses. One of these days the A. V. M. A. is going to repudiate the gratuitous slap which it took at anti-rabies vaccination at the Washington meeting, and when that occurs some newspaper columnist, who is also a rabid anti-vaccinationist, may take the A. V. M. A. dog food approval project for a ride that will not make pleasant reading for veterinarians. The whole program should be reëxamined by the House of Representatives at the Indianapolis meeting.

The Practitioners' Association

One of several organizations that will hold meetings in Indianapolis during the annual meeting of the A. V. M. A. is the American Society of Veterinary Therapy or, as it is almost universally called, the "Practitioners' Association." The room, day and hour of the meeting will be announced in the regular A. V. M. A. program. The officers invite all veterinary practitioners, whether or not they are members, to attend this meeting.

The practitioners' association is the newest among national veterinary associations, having been organized only one year ago last March. It represents the largest and most important branch of the profession—the practitioners who comprise approximately 65% of the whole number of veterinarians in the country. In membership gains the new association has made a remarkable showing. Within 12 months of its organization it had attained a paid up membership of 683. It took the A. V. M. A. right at 50 years to attain this membership. The U. S. Live Stock Sanitary Association in two score years reached but half this number. The National Association of B. A. I. Veterinarians, after 20 years of successful organization, has fewer members than the practitioners' association has garnered in a single year, while the small animal practitioners' association in five years gained but a tenth of this number.

J. C. Flynn, Secretary of the Practitioners' Association, 3026 Main St., Kansas City, Mo.; President of the A.V.M.A. 1935-36

No one can survey the career of veterinary medicine in this country appraisingly without becoming convinced that a large, active, well directed association of general practitioners working within and through the A. V. M. A. would be highly advantageous not only to the branch of the profession they represent but to the whole profession and to the national association also. The associations of veterinary college faculties, of those engaged in veterinary research, of the live stock sanitary forces of the Bureau veterinarians, and of the small animal practitioners each in turn have brought about improvements in their particular fields that have redounded to the advantage of the profession as a whole. The practitioners' association has a greater opportunity to improve its own field and veterinary medicine as a whole than any of the other branches of the profession had when it was organized or than it has now; not only because the field is larger and more important, but because it is the field in which the most needs doing.

No one could truthfully gainsay that little, if anything, of a sustained, systematic nature was ever done for the protection of clinical veterinary medicine by the national association. Its work, although always more or less hit-and-miss, is not discounted so far

Practitioners have for years felt the need of an organization to represent their particular interests in the field of veterinary medicine. Each of the other branches of the veterinary profession has its own organization with the result that in the A. V. M. A. these groups, although much smaller numerically than the unorganized practitioner group, wield a far greater influence; in fact the practitioners, the backbone of the profession, can scarcely make themselves heard or felt in our national meetings and their specific problems are neglected

The Practitioners' Association is truly representative of the practicing veterinarian. It has grown and continues to grow amazingly, a circumstance which presages a useful future

S. L. STEWART.

Olathe, Kans.

as the general promotion of the profession is concerned. In the matter of studying the problems of the practitioners, however, its promotional program has been 100% void—no one has ever championed the practitioners' cause except in the beautiful language of presidential addresses which has always been quickly forgotten. Such platitudes as "The practitioner is the bulwark of the profession" have been doled out *ad nauseam* at so many opening sessions, and so little has ever been done to justify the taffy, that the formation of a practitioners' society became inevitable.

The reasons for the neglect of clinical veterinary medicine are numerous, but the principal one is lack of leadership due in the main to geographic obstacles. Until within recent years the organization of the national association placed its control wholly in the hands of those who attended its annual meetings and almost as effectively in the hands of those who attended those meetings year after year over a long period. This included precious few general practitioners. The present organization of the A. V. M. A. was designed to place the control of the organization largely in the membership at large. Whether or not a member attends the annual meetings he can have a voice in the selection of the officers and indirectly a voice in the policies of the A. V. M. A. Strangely, practitioners have not availed themselves of this opportunity which the association has tendered them. A little investigation of votes cast for executive board members revealed fewer than one in seven cast by practitioners in the nominat-

I joined the American Society of Veterinary Therapy as soon as I learned that a group of practitioners had banded together to work out their own problems—the problems of the practicing veterinarians. I believe that much good will result from it

We should be more active in our state associations. We should have someone looking after our interests in legislation, city, state and national. I believe we should have a committee of practitioners to confer with a like committee from the commercial houses, the B. A. I. and serum producers when regulations are to be put into force which vitally concern us. This would prevent much misunderstanding and friction. This can and should be done, and this association is working to that end

If we as practitioners do not assert ourselves, we must expect infringements on our practice. We cannot accomplish much single handed, we must cooperate. The least we can do is to support this important work of earnest, hard working men who have started the only cooperative movement devoted entirely to our problems. If all public spirited practitioners will get behind this association, outstanding achievements will result from it in the next few years. I am a firm believer in the practitioners' association

J. E. WEINMAN.

Lincoln, Nebr.

ing ballot and always less than a majority in the election. Since the number of general practitioners in the House of Representatives is small, one may assume that practitioners have been equally indifferent to the selection of this, the legislative body of the A. V. M. A. Possibly that assumption is not justified; the writer possesses no definite information on the matter.

The foregoing appears to delineate the problem confronting the newly organized practitioners' association—the problem that will determine its usefulness or its inutility, its success or failure. The problem is one of educating the private practitioner of veterinary medicine to take a greater interest and give more active support to organized veterinary medicine, through which all progress and improvement in the profession is achieved. This increased interest and support should begin with the state associations and must include not only paying dues and attending meetings, but work on committees, contributing to the program and to the discussions out of which come policies. And so far as it be practical, the same support must extend to the national association.

It is preposterous to say, as has been said, that the A. V. M. A. is antagonistic to the practitioner. The A. V. M. A. doesn't know the practitioners' problems because he hasn't told it what those problems are. It hasn't done much of anything for him because he has not said what he wants done. It has seldom elected him to a place on its executive board because it is not often that

During such stressful periods as the present, the importance of organization comes to the fore. No unorganized army or defense effort can make substantial progress, win battles or help win a war without constant backings, turnings and stallings

These principles were recognized by a group of progressive veterinary practitioners who decided upon the formation of the American Society of Veterinary Therapy, an organization of practicing veterinarians In our councils we have long recognized the prime importance of an improved service for the live stock industry. We recognize the practitioner as the only logical individual through whom the benefits of improved methods of therapy can be extended to our live stock. Only through him can the fine work of our research staffs be made available where it is needed. Only through him can the official disease control measures be carried out on a wide scale. Only through him can the preparations of those engaged in commercial lines be applied intelligently to the uses for which they are intended

Efficient production of live stock and dairy products for food purposes is dependent upon the health of our domestic animals. We practitioners are charged with the maintenance of that health. The demand of our government for more meat production, more dairy production and more egg production will not be met without the services of a highly skilled personnel for the control of disease and that personnel must be on the job all the time, day and night, and on the spot where the animals and the disease are located. Food is a munition of the first magnitude, in both peace and war. Malnutrition from lack of proper food lowers the fighting efficiency of an army and the productive capacity of a civilian population

A profession justifies its existence only as it serves a specific function in the commonwealth. Our branch of the veterinary profession—practitioners—is charged with the prevention of disease in our herds and flocks and the conservation of sick domestic animals. We can accomplish this far more effectively by coordinated efforts than by individual struggle. We need to help one another with our problems. This is the reason for the new practitioners association. This is why I support it

GEO. A. HAWTHORNE.
Clarinda, Iowa.

he has taken the trouble to vote, and probably his rarity among the members of the House of Representatives is due to the same indifference.

The A. V. M. A. will profit tremendously from an awakened interest of practitioners in organized veterinary medicine, and not alone in members, although that is important. It will profit even more in wisdom and influence. If the American Society of Veterinary Therapy (that name should be changed; this writer always has to hunt up one of the society's letterheads to recall it) can awaken an interest in and enthusiasm for organized veterinary medicine among the general run of practitioners and can enlist their active and continuous support of organized veterinary medicine in the state and in the nation, it will have deserved well of the whole profession. This has been tried repeatedly without success. However, it has never before been tried on the large scale now attempted; nor has comparable success ever before attended the initial effort. All farseeing veterinarians will lend the new practitioners' organization every possible encouragement.

The American Society of Veterinary Therapy, the National Practitioners' Association, which was organized a few months ago, is one that every veterinarian in general practice should join and support

First, it is an organization for the practitioners, the men who are really in the field and who contact the producers of the nation's food supply; the men who advise as to the breeding, feeding and care of our live stock. The practitioners in the field, the veterinarians who have to rough it, should join together and lay their plans to accomplish the most good. Our cities and armies cannot be fed if we practitioners "lay down" on our jobs

The history of the control or eradication of Texas fever, foot-and-mouth disease, anthrax, pleuro-pneumonia, sheep scabies, tuberculosis, glanders, hog cholera and numerous diseases of poultry, all of which have been successfully handled, shows that the practitioners are the main stay in disease control; their diligence, thorough and comprehensive service and long hours make the success of the live stock sanitary officials possible

S. E. HERSHEY.

Charleston, W. Va.

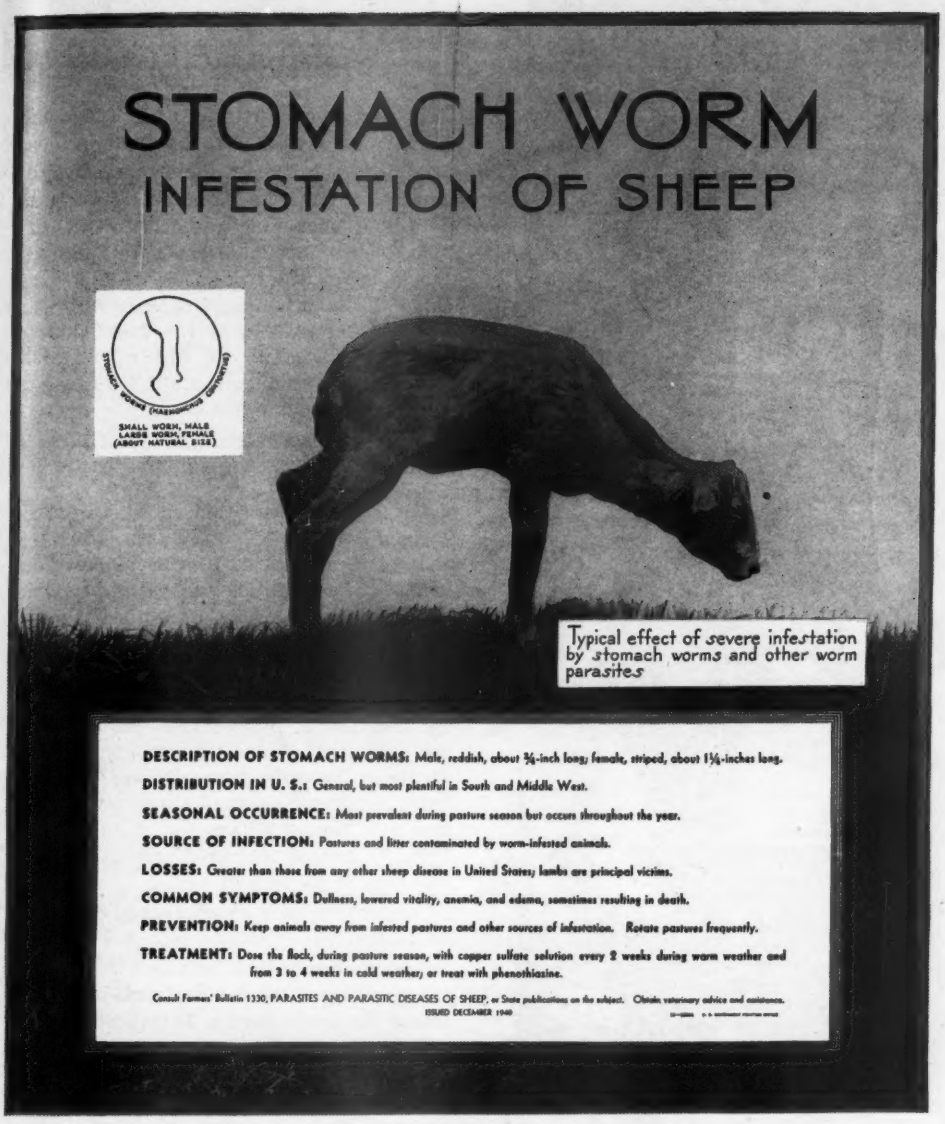
The average working day of the veterinary practitioner is of necessity very long. He is obliged to spend the greater share of this time at his work. He has little opportunity for conferences with other groups of the profession. This I think is the principal reason why the "corner stone" of the veterinary profession—the practitioners, do not wield sufficient influence when important policies are being formulated

Veterinary practitioners constitute a majority of all groups charged with the health of the animals, yet very few have a voice in preparing regulatory measures

If for no other reason than this, the practitioners of the American Veterinary Medical Association should have a group organization where their ideas may be discussed and unified, then presented for consideration along with the ideas of other groups within the profession. Group organizations in the A.V.M.A. are timely and helpful. They should be encouraged

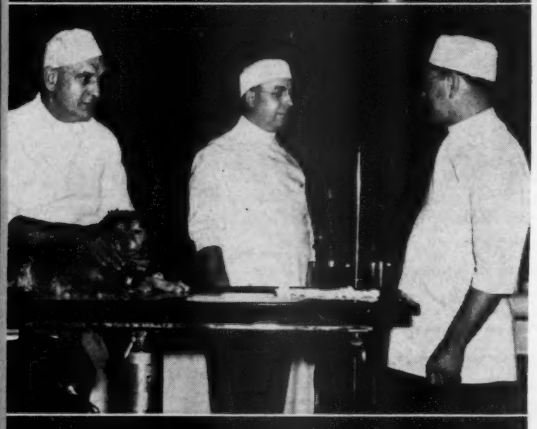
CHARLES W. BOWER.

Topeka, Kans.


STOMACH WORM
INFESTATION OF SHEEP
STOMACH WORMS (HAEMONCHUS CONTORTUS)
SMALL WORM, MALE
LARGE WORM, FEMALE
(ABOUT NATURAL SIZE)
Typical effect of severe infestation by stomach worms and other worm parasites
DESCRIPTION OF STOMACH WORMS: Male, reddish, about ¾-inch long; female, striped, about 1¼-inches long.
DISTRIBUTION IN U. S.: General, but most plentiful in South and Middle West.
SEASONAL OCCURRENCE: Most prevalent during pasture season but occurs throughout the year.
SOURCE OF INFECTION: Pastures and litter contaminated by worm-infested animals.
LOSSES: Greater than those from any other sheep disease in United States; lambs are principal victims.
COMMON SYMPTOMS: Dullness, lowered vitality, anemia, and edema, sometimes resulting in death.
PREVENTION: Keep animals away from infested pastures and other sources of infestation. Rotate pastures frequently.
TREATMENT: Dose the flock, during pasture season, with copper sulfate solution every 2 weeks during warm weather and from 3 to 4 weeks in cold weather; or treat with phenothiazine.
Consult Farmers' Bulletin 1330, PARASITES AND PARASITIC DISEASES OF SHEEP, or State publications on the subject. Obtain veterinary advice and assistance.
ISSUED DECEMBER 1940

"Permanent Pastures Perpetuate Parasites"

"Once animals are under fence our parasite cycle begins; increased land values call for more and better stock per acre; more stock per acre means more manure per acre; more manure per acre means more parasite material per acre; more parasite material means greater certainty of infection and greater infection per animal; and greater infection per animal means more worm material for any given amount of manure; and this means more infection per acre, and so on."—*Maurice C. Hall*, 1928.



Veterinarians of

The clinic was an interesting and much appreciated feature of the 57th Annual Meeting of the Indiana Veterinary Medical Association, held in Indianapolis, January 14-16, 1941. The clinic was held in the ballroom of the headquarters hotel and although scheduled from 7:00 to 9:00 p.m., lasted till near midnight. The illustrations herewith show some of the highlights of this excellent clinic, at which Dr. E. K. Sales of the Michigan State College was guest diagnostician and surgeon.

In the top illustration a group of Indiana veterinarians is shown watching the clinic.

■ The second illustration shows an operation for otorrhea. Those shown, from left to right, are: Drs. C. P. Wilson, Lawrence (whose patient the dog was); H. J. Magrane, Mishawaka; H. Meade Hamilton, Muncie; and E. K. Sales at the microphone.

This dog had been treated for chronic ear canker for many months, practically all the recognized treatments for canker having been used at one time or another, but all the while grew gradually worse. Doctor Sales performed the well known "V" operation and Doctor Wilson has since reported that the animal recovered promptly and at this time is sound and well.

Concerning such cases, Doctor Sales said in substance:

Various degrees of inflammation involving portions of the internal part of the external ear are common. They produce conditions we diagnose as otorrhea or otitis externa.

Cases of otitis caused by foreign objects lodged in the ear canal have responded favorably to treatment after removal of the object. The same has been true of those cases caused by the presence of ear mites, in most instances. It is worth noting that ear mites are present in most cases of otitis involving cats.

Dean Brumley's treatment for ear mites has produced good results for us and is especially useful for those cases where the owner complains of an offensive odor in

Indiana Hold 57th Annual Meeting

connection with the infestation. It consists of:

In the first place, the removal of all dirt and cerumen which has accumulated. This is best accomplished by taking pledgets of cotton or gauze on a dressing forceps, saturating them in ether, and thoroughly swabbing out the whole canal until all the débris is removed. In this cleansing process all of the small interstices between the swollen masses should be opened and swabbed. The ether solution is necessary in order to dissolve the cerumen. With the scissors cut away all superfluous hair in the canal. Dry cotton or gauze should then be used in making the surface perfectly dry and also to remove any excess of ether which might be retained. An antiseptic powder (boric acid; zeriform; tannoform) is worked down carefully into the canal to produce antiseptic action and to prevent the secretions adhering to the skin surface. This treatment should be applied daily until the inflammation and swelling subside.

Canex and pellitol have both proved of value in these cases. For those cases of otitis due to infection by such organisms as *Staphylococcus albus,* hemolytic *Staphylococcus albus* and *Streptococcus viridans,* etc., I have no dependable treatment to offer. Many of these cases respond to one treatment or another, many other cases do not. Whatever treatment one decides to use, unnecessary probing and swabbing should be avoided. We often prefer to irrigate the canal with warm non-irritating antiseptic solutions, especially in cases where other methods of cleaning are painful and appear to aggravate the condition.

Operations to establish drainage are well worth trying in cases such as this that fail to respond to other treatment, if the owner will consent.

Many cases of otitis are accompanied by the formation of ulcers. In most instances, these occur on or near the roof of the canal. Cauterizing them lightly with silver nitrate often causes them to heal rather quickly.

■ The third illustration shows a mongrel presented for demonstration of an entropion operation. The operation was performed under local anesthesia, the chief novelty of it being that the operation was performed without clipping the hair from the area. Entropion seldom develops in other than young dogs and they do well operated upon in this manner. They can be entered in shows much sooner after the operation if the hair is not clipped than if it is. Owners appreciate this highly.

Those shown in the illustration are: E. K. Sales (center), East Lansing, Mich.; assistants, H. J. Magrane (left), Mishawaka, and H. Meade Hamilton, Muncie.

■ The lower illustration shows the castration of a dog. Surgeon, E .K. Sales; assistants, Paul J. White (left), Indianapolis; H. J. Magrane and H. Meade Hamilton (right).

An area covering the scrotum and for an inch or two forward was closely clipped of all hair. This area was then washed with soap and water, followed by 70% alcohol, and three to 5cc of procaine solution was injected along the cord of each testicle and massaged well.

As soon as the anesthetic had taken effect, the right testicle was pushed ahead of the scrotum and an incision just large enough to permit the testicle to pass through it was made near the median line.

The testicle was pulled well out and the cords securely tied with No, 0 chromic catgut. The testicle was then clipped off with scissors. The opposite testicle was then pushed forward so that it bulged into the same opening, the median raphe was incised, and the testicle was removed through the same skin opening in the same manner as the first testicle.

The skin opening was then tightly closed by suturing with No. 0 catgut and the patient was ready to send home.

This method of castration of the dog possesses the following advantages: The patient can ride home in the owner's car immediately after the operation, in most cases, without spotting the upholstery of the car and can be allowed the run of the house as usual without damage to rugs or furniture. No aftercare has ever been required. Patients kept and observed for several days in the hospital show only a moderate fullness in the scrotum for two or three days and then all swelling subsides without any discharge.

Lymphoblastoma in Dairy Cattle*

By L. S. STARR, D.V.M., M.S., Ph.D.
and TYLER YOUNG, B.S., M.S.
Auburn, Alabama

LYMPHOBLASTOMA is a tumor-like disease which occurs in several species of animals, notably cattle, dogs and man. It is characterized by the formation of tumorous masses in most of the organs of the body; being confined almost entirely to the lymphoid tissue of the organs. The disease progresses quite rapidly and is usually fatal. Its rapid growth and wide distribution throughout the body suggests a malignant neoplasm, but whether it is a hyperplasia of lymphoid tissues already present or whether the new growths are the result of metastasis has not been definitely determined.

There is considerable confusion concerning the correct classification and nomenclature of the condition. Boyd includes leucemia, lymphosarcoma and Hodgkin's disease, all of which bear considerable histopathological similarity, under the general classification of malignant lymphoma. Leucemia is subdivided into leucemic leucemia, in which there is an increase in the lymphocytes in the blood, and aleucemic leucemia in which there is little or no increase of lymphocytes in the blood stream. The disease described in this paper belongs in the aleucemic leucemia group and is generally designated as lymphoblastoma.

Occurrence

Lymphoblastoma is comparatively common in adult cattle, particularly among the dairy breeds. The annual loss of several head of mature animals is not uncommon in many herds.

Etiology

The etiology of lymphoblastoma has not been definitely established. Various theories have been advanced but none have been proved. It may be a simple hyperplasia due to some stimulus such as a foreign body, the continued irritation causing some tissue reaction or secretion which stimulates cell division. The primary stimulus might be local and the general distribution of the hyperplastic nodules embolic in origin.

It is possible that it may be due to a virus, although there is no evidence to support this, other than the fact that a somewhat similar condition in the chicken is known to be due to a virus.

The fact that the disease appears quite frequently in certain families of cattle indicates that a genetic factor may be involved.

The frequency of clinical manifestations of disease at or about the termination of pregnancy may indicate an association with a disturbance of the endocrine glands. If so, it appears likely that the endocrine disturbance acts as an accelerating factor rather than as a primary cause of the condition.

Symptoms

The most common symptoms observed are those of a typical case of indigestion. There is complete absence of rumination with loss of appetite, and cessation of milk flow.. In many instances palpation reveals no enlargement of the superficial lymph nodes; in some cases they are not only palpable but visible. With appropriate treatment for indigestion, recovery is usually prompt but temporary, and one is called again in a few days because of a recurrence. From this time on the animal becomes progressively worse. Subcutaneous edema is common, but not constant. There is an irregular appetite or complete anorexia, rapid loss of flesh, and cessation of milk flow; the animal becomes comatose and dies. In herds in which lymphoblastoma is known to be present recurrent cases of indigestion should be regarded with suspicion. Even in early cases, total and differential blood counts are of material assistance in the diagnosis.

We have observed a few atypical cases, most of which were not diagnosed as lymphoblastoma until necropsy revealed the characteristic tumor-like growths. Some of these cases are of considerable interest.

Case 1.—A Jersey cow in apparently good health delivered a normal calf without as-

*Experiment Station Project, Department of Animal Pathology, Virginia Polytechnic Institute, Blacksburg, Virginia.

sistance. About three weeks later she developed what appeared to be a typical case of indigestion, was treated for this condition, and was well two days later. Three days later she was reported in a serious condition. Examination revealed the cow standing in the manger with her shoulders elevated and her head extended, respiration rapid, temperature 104.2° F. and a rapid, pounding heart beat. There was a very extensive subcutaneous edema from the rami of the mandible to the mammary gland. She was in very great distress and died the next day, six days after onset of the illness. Necropsy revealed a cylindrical tumor mass approximately nine by 30 inches, involving the rumen, reticulum, omasum, abomasum, and duodenum. All the abdominal lymph glands were greatly enlarged. Large tumorous masses were present in the liver, kidneys, spleen, uterine wall and intestines. Several small growths were present on the myocardium and upon incising the heart a large tumorous mass was found which practically filled the right auricle, seriously impeding return flow of blood from the common vena cava. The attack of indigestion was undoubtedly due to the tumorous mass involving the stomachs and duodenum, but the immediate cause of death was the mass in the right auricle.

Case 2.—A six-year-old Guernsey bull began to show knuckling of both rear pasterns. The condition progressed until the animal became completely paralyzed in the rear quarters and was destroyed. Necropsy revealed typical tumorous masses involving most of the lymphoid structures of the abdominal cavity and a small mass in the spinal canal which had caused pressure on the cord resulting in the paralysis.

Case 3.—A Jersey cow began to show inappetence, rough hair coat, cessation of milk flow and loss of flesh for no apparent reason. In a few days it was noted that her eyeballs were unusually prominent. All the symptoms became progressively worse until she became prostrate and was destroyed. A rather unusual condition was present in that the eyeballs protruded to such an extent that the eyelids could no longer wash the eyes.

Necropsy revealed typical tumorous masses involving the lymphoid structures of the body and a small mass in the cranium which was causing pressure on the optic nerves, resulting in protrusion of the eyeballs. This animal had the typical appearance of extreme exophthalmic goiter in man.

In the course of necropsies over a period of years, we have observed that the primary tumor process appears to be on or near the rumen or reticulum. These organs in cattle are also the most common sites of penetration of foreign bodies such as wires and nails, which might act as irritants to the tissues, causing a local hyperplasia. Chronic irritants are believed to be one of the common causes of tumor formation of various kinds.

We have observed that the cases occurred largely in two families, indicating a possible genetic relationship.

Experimental Procedure

The project was set up to include three phases:

1. Production of tumors following artificial irritation.
2. Transmission by means of transplants of tumor tissue.
3. Investigation of genetic history.

Production of Tumors Following Artificial Irritation

Four adult dairy cows were selected for this phase of the experiment.

Animal No. 1.—Laparotomy was performed under local anesthesia, April 28, 1936, and a six-inch piece of bailing wire anchored in the wall of the rumen. The effects on the blood picture and general condition are shown in Table I.

TABLE I.

Date	R.B.C.	W.B.C.	Hem. %	Differential P.	L.	E.	M.	B.	Remarks
4/28/36	6,122,000	12,550	72%	37	50	8	2	3	Normal
7/29/36	5,900,000	25,120	75%	66	22	10	0	2	"
1/15/37	6,322,000	21,500	76%	34	52	12	0	2	"
8/18/37	6,220,000	18,520	76%	22	55	18	5	0	"

This animal showed a decrease in erythrocytes and an increase in leucocytes three months following insertion of the foreign body, the blood picture being quite typical of that observed when an abscess is present. This abscess if present and the wire were probably absorbed without producing tumor tissue. The animal, in apparently normal condition, was sold for slaughter in January, 1939, and no necropsy was performed.

Animals Nos. 2, 3, 4.—A trocar was inserted in the rumen of these animals and two pieces of wire six inches long, two paper clips and two finishing nails passed through the canula

into the rumen of each. The results are shown in Table II.

Typical cases of traumatic pericarditis were produced in these three cows following insertion of foreign bodies into the rumen. There was considerable variation in the length of time between insertion of the foreign bodies into the rumen and death. In cow No. 2 this interval was 41 days, No. 3, 271 days, and No. 4 approximately 1300 days.

No pathological changes indicative of tumor formation were observed at necropsy of any of these animals.

TABLE II.

Date	R.B.C.	W.B.C.	Hemo. %	Differential P.	L.	E.	M.	B.	Remarks
Animal No. 2									
5/14/36	6,210,000	8,540	74%	35	42	6	10	7	Apparently healthy
6/1/36	5,840,000	16,600	70%	55	30	5	9	1	Anorexia, rough hair coat
6/5/36	5,470,000	22,700	65%	60	25	3	9	3	Condition more critical
6/10/36									Edema, jugular pulse, hydropericardium
6/24/36									Died. Necropsy—traumatic pericarditis
Animal No. 3									
5/14/36	5,630,000	12,130	75%	42	43	1	2	1	Apparently healthy
7/22/36	5,920,000	16,120	60%	45	47	5	3	0	Apparently healthy
12/14/36	5,435,000	16,520	62%		Not recorded				Anorexia
1/13/37	3,120,000	28,000	32%		Not recorded				Condition critical
1/23/37	2,860,000	46,120	*		Not recorded				Condition critical
2/1/37	3,300,000	36,200	*		Not recorded				Condition critical
2/8/37	3,240,000	41,000	*		Not recorded				Comatose condition
2/9/37									Destroyed, Necropsy revealed pericarditis
Animal No. 4									
5/14/36	5,180,000	11,880	60%	53	39	7	0	1	Normal
7/22/36	5,130,000	12,320	54%	57	35	6	1	2	Normal
8/18/37	5,220,000	8,120	56%	26	49	21	4	0	Normal
Dec./39									Died. Necropsy revealed traumatic pericaditis

*Too low to read.

Injections with Tar and Lard

Three cows were given weekly intramuscular injections of a 4% suspension of tar in lard over a period of six months. The injections were started in February and continued through June, 1936. The blood pictures during this period remained within the normal range.

One animal died in May, 1938. The muscular tissue at the sites of injection of the tar suspensions was discolored but there was no indication of hyperplasia of the lymphoid tissue. A second animal, sold for slaughter was apparently normal.

The third animal died in November 1940, of generalized lymphoblastoma. It should be noted that because of the limited number of animals employed, it is impossible to determine whether the lymphoblastoma in this case was induced by the tar injections or arose from other causes.

Transmission by Means of Transplantation of Tumor Tissue

Experiment 1.—A Holstein cow with generalized lymphoblastoma was destroyed December 20, 1936. Some of this tissue was immediately shredded and frozen solid in order to hold the tissue until a newborn, closely related male calf was available. December 26, 1936, tissue filtrate was injected in a newborn Holstein calf as follows: ½cc intratesticularly, 1cc intramuscularly and 1cc subcutaneously. This calf was sacrificed 13 months after these injections. No lesions suggestive of hyperplasia of lymphoid tissue were observed at necropsy.

Experiment 2.—Lymphoblastoma tissue was obtained from a Jersey cow within ten minutes after death. Using a ten-gauge needle, small masses of this tissue were injected intratesticularly, subcutaneously and intramuscularly into a newborn calf. This animal was sacrificed after 11 months. No lesions suggestive of hyperplasia of lymphoid tissue were observed at necropsy.

Thus in one instance a filtrate from frozen tissue was employed, in the other fresh tissue clumps were injected. Negative results were obtained in both instances.

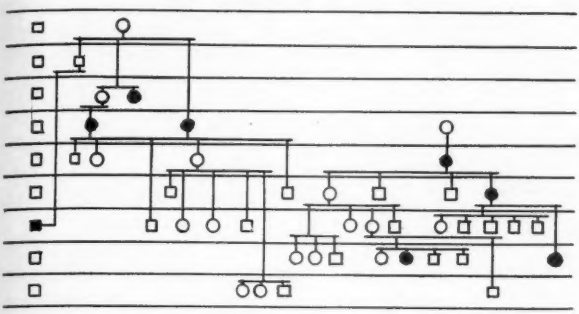
Genetic Relationship

A study of the necropsy and pedigree records revealed that the majority of the cases of tumors had occurred in two families of cattle, which indicated the possibility that genetic factors were involved.

The genetic relationship of the animals

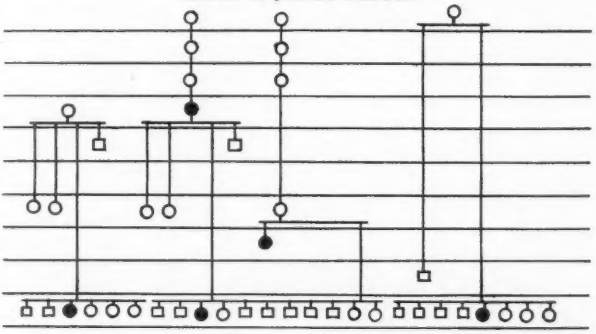
that have died in the college herd was assembled in chart form as shown in Charts I and II. Although the number of animals concerned is too small for definite conclusions to be drawn, it is of interest to note that most of the affected animals of each family were fairly closely related.

CHART I. HOLSTEIN RECORD



In the Holstein breed, Chart I, one apparently normal cow had two daughters, a granddaughter and a grandson which died of lymphoblastoma. Another cow, her daughter, granddaughter, and great granddaughter were similarly affected. In the Jersey breed, Chart II, the cases occurred in less closely related families than in the Holstein group.

CHART II. JERSEY RECORD



Discussion and Conclusions

Lymphoblastoma is relatively common in some dairy herds and since it is invariably fatal, it results in a considerable economic loss. It is apparent that an excitant of some nature acts as a stimulating factor causing an extremely rapid hyperplasia of the lymphoid tissue of the body. Whether this factor is a virus, some foreign irritant, or of genetic nature is not definitely known at present.

Typical attacks of traumatic pericarditis were produced in three cows under controlled conditions, but no hyperplasia of lymphoid tissue was stimulated in any instance. The leucocyte picture was typical of that of a foreign body with tissue infection and not of lymphoblastoma. Another animal, in which the wire was fastened to the wall of the rumen, showed a blood picture which indicated abscess formation, later returning to normal. She was sold for slaughter 23 months after the operation with no indication of tumor formation.

Three cows received weekly intramuscular injections of a 4% suspension of tar and lard at weekly intervals over a period of six months. One animal died of generalized lymphoblastoma approximately three years after the injections were started; the other two remained healthy until sold for slaughter. The positive results in one of three animals are not conclusive, as this may have been a case of tumor formation from other causes.

Attempts to transit the condition by direct implantation of fresh tumor masses in one male calf and by injection of tissue filtrate in another calf were unsuccessful. Since only two animals were used, these negative results are inconclusive. Further work should be done to determine definitely whether or not a virus is involved.

There is some evidence of a possible genetic factor. In the Holstein breed all the cases occurred in two families. The incidence in certain families was not so pronounced in the Jersey cattle. Again, the number of cases is too small to justify drawing definite conclusions.

The History and Distribution of Equine Infectious Anemia in the United States

By C. D. STEIN, Washington, D. C.
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EQUINE infectious anemia, on account of its widespread distribution and its insidious nature, has become a problem of serious concern to owners of horses and mules in all parts of the world, and until effective methods for its detection and control are perfected it will continue to be one of the more important diseases of solipeds.

The disease which was first reported from Europe in 1843 now has a world-wide distribution, having been reported from various parts of Europe, Asia, Africa, and North America, and recently from Venezuela, South America, by Kubes[1].

Dobberstein and Hemmert-Halswick,[2] in a recent article dealing with world distribution of infectious anemia from a geographical and geological standpoint, reported that in addition to Canada and the United States the disease exists in certain well-defined areas in the following countries: Germany, France, Switzerland, Sweden, Norway, Finland, Yugoslavia, Hungary, Union of Soviet Socialist Republics, Japan, and sections of northern Africa. They also pointed out that neither the geological structure nor the water courses of a given region appear to have any outstanding significance on the dissemination of the disease.

Early History of the Disease in North America

Although there appears to be little doubt that the disease has existed in the United States for more than 50 years, it is uncertain when the malady was first identified as a specific equine disease in North America. It appears to have been recognized as a distinct disease of horses about 1880 along the valley of the Red River in the Province of Manitoba, Canada, but it was not referred to as swamp fever at that time. However, even previous to this time a disease in horses resembling swamp fever had been observed in western Canada.

Panisett[3], in a recent article dealing with the history and distribution of the disease in Canada, mentions that the first allusion to the disease in Canada appears to have been made by Kane in 1855, who described a disease in horses near Winnipeg with a high rate of mortality and which he attributed to parasitic infestation. First definite mention of the disease in Canada was made by Rutherford[4] in 1888, who described a disease in horses closely resembling infectious anemia which he believed to be a protozoan infection and which he called American surra. Rutherford reported observing cases of the disease as early as 1884 and indicated that it existed as early as 1880. Rutherford[5] later reported further observations on the disease, calling it swamp fever. However, it appears that Torrance[6] was the first definitely to recognize and describe the malady as a specific disease of horses. He reported that veterinarians in the Province of Manitoba in 1881 and 1882 observed a disease in horses having all the characteristics of infectious anemia, and in 1902 made a report describing the disease in detail and naming it malarial fever of horses. Torrance[7, 8] made a detailed

[1] Kubes, Vladimir, 1939. Estudio acerca de la existencia de la anemia infecciosa de los equinos en la America del sur. Su presencia en Venezuela y confusion con la tripanosomiasio caballar—las llamadas "peste boba" y derrengadera E E U U De Venezuela, Ministerio De Agricultura y Cria, Caracas.

[2] Dobberstein, J. and Hemmert-Halswick, G., 1940, Die verbreitung der infektiosen Anamie nach geographischen und geologischen Gesichtspunkten, Zeitschr. f. Infektkr., paras. Krankht. u. Hygiene der Haust. Bd. 57, Hft. 1, S. 55-74 (Distribution of infectious anemia from the geographic and geologic viewpoint).

[3] Panisset, M., 1938. Historique et repartition geographique de l'anemie infectieuse des equides au Canada. Cah. Med. vet. V. 7, pp. 136-138. *Vet. Bul.* V. 8, No. 3, p. 153.

[4] Rutherford, J. G., 1888. Equine surra, *Vet. Rec.* Vol. 1.

[5] Rutherford. J. G., 1903. Swamp fever, Rep. of the Minister of Agr. for the Dom. of Canada for the year ended Oct. 31, 1902, p. 85, Ottawa.

[6] Torrance, F. W., 1903. Report on "swamp fever" in horses. Rept. of the Minister of Agr. for the Dom. of Canada for the year ending Oct. 31, 1902, p. 85, Ottawa.

[7] Torrance, F. W., 1903, 1904. Report on swamp fever in horses, Ann. Repts. 1903 and 1904. Dept. Agr., Canada.

[8] Torrance, F. W., 1909. Report of swamp fever investigations. Report of the Vet. Dir. Gen. and Live Stock Comm., 1906-1908, p. 71, Ottawa,.

investigation of the disease between 1902 and 1906, the results of which were published in additional reports.

Taylor[9] in 1899 also reported the disease in Manitoba and referred to it as malaria fever. Studies of the disease and early reports of its existence in Canada were also made by Acres,[10] Ballah,[11] Charlton,[12] McGilvray,[13] and by Todd and Wolback.[14] The last mentioned authors made an extensive investigation of the malady.

Panisett[3], in tracing the spread of the disease in Canada, pointed out that the disease appears to have spread from Manitoba to the west until held up by the Rocky Mountains and to the east through Ontario to Quebec.

Early History of the Disease in the United States

Although the disease appears to have spread along the valley of the Red River from the Province of Manitoba into some of the northwest territories and was first definitely recognized in Minnesota, it is highly probable, in view of its obscure nature, that it may have existed unrecognized in these and other localities many years prior to its identification. It is worthy of note that subsequent to the definite recognition of the disease in Manitoba and Minnesota, it was widely identified in different sections of both Canada and the United States.

The first recognized outbreaks in the United States occurred chiefly in low-lying, poorly-drained, marshy sections of the country. For example, outbreaks of the disease were first observed along the valley of the Red River in Minnesota and North Dakota; in the valleys of the Big Horn, Laramie, and Little Laramie Rivers in Wyoming; in the valley of the Platte River in Nebraska, Wyoming, and Colorado; in the Mississippi Delta of Louisiana, Mississippi, and Arkansas; and along the low-lying wet coastal plains of the Gulf of Mexico in Texas. Although the disease occurs in rolling country, well-wooded sections, and at high altitudes, new outbreaks for the most part have been reported principally from low-lying poorly-drained areas. However, outbreaks may and do occur in any locality regardless of altitude or drainage when infected animals are brought into non-infected territory and circumstances are favorable for transmission to susceptible horses.

First Reports of the Disease in the United States

Schroeder,[15] a veterinarian of the Bureau of Animal Industry in 1892 reported the investigation of two horse diseases in the West, the first the so-called bottom disease affecting only horses and occurring along the bottom lands of the Missouri River in southeastern South Dakota, northeastern Nebraska, western Iowa, and the second, a peculiar affection among range horses south of the North Platte River in Wyoming, the Wyoming disease making its appearance in the latter half of summer and continuing until October, causing great annual losses. While certain aspects concerning both of these diseases as described by Schroeder are somewhat similar to swamp fever, it is impossible to state with certainty whether or not they were identical conditions.

Watson[16] in 1896 reported that a disease, which he called equine relapsing fever, with symptoms identical with infectious anemia, was observed in horses in the State of Wisconsin as early as 1888.

Mack[17] reported an outbreak of an anemic disease in horses in Nevada during 1906, 1907, and 1908, which he believed to be of a specific infectious nature and which was later identified as infectious anemia. Ranchers in this territory were of the opinion that the disease had existed for 15 to 20 years previous to 1906.

[9] Taylor, 1899. A case of malarial fever, *The Vet. Jnl.*, Vol. 49, p. 1, London.
[10] Acres, G. H., 1909. Infectious anemia or swamp fever, *Amer. Vet. Rev.*, Vol. XXXIV, p. 525, New York.
[11] Ballah, 1908. Report on "swamp fever," Ann. Rept. of the Dept. of Agr. of the Prov. of Saskatchewan 1907, p. 223, Regina.
[12] Charlton, G. A., 1907. Swamp fever, Ann. Rept. of the Dept. of Agr. of the Prov. of Saskatchewan 1906, p. 146, Regina.
[13] McGilvray, C. D., 1910. Rarer forms of fever in horses, Proc. of the Vet. Assn. of Manitoba, 1910, p. 23, Winnipeg.
[14] Todd, J. T. and Wolbach, S. B., 1911. The swamp fever of horses, The *Jnl. of Med. Res.*, XXIV, p. 213, Boston.
[15] Schroeder, E. C., 1891-1892. Diseases of horses in the West, 8th and 9th Ann. Repts. of the Bur. An. Ind. for the years 1891 and 1892, pp. 104, 371-374.
[16] Watson, W. E., 1896. Equine relapsing fever, *Amer. Vet. Rev.*, Vol. XX, p. 196, New York.
[17] Mack, W. B., 1909. A study of a serious anemic disease among horses, *Amer. Vet. Rev.*, XXXVI, p. 222, New York.

Scott[18] who studied the disease in Wyoming, expressed the opinion that infectious anemia existed in that state many years before any records were made concerning it. He mentions an extensive outbreak that occurred in 1901 and 1902, and points out that the older ranchmen in the valley of the Little Laramie River reported that the same disease was prevalent in the early nineties.

Following the definite recognition of the disease in Minnesota in 1903 by Brimhall, Wesbrook, and Bracken,[19] and in 1905 by Beebe,[20] it was reported from Nebraska in 1906 by Peters,[21] from North Dakota in 1907 by Van Es,[22] from Texas in 1908 by Francis and Marsteller,[23] from Louisiana in 1909 by Flower,[24] from Wyoming and Colorado in 1909 by Whitehouse,[25] from Oklahoma, Kansas, and Missouri in 1910 by Kinsley,[26] from Washington in 1910 by Thatcher,[27] from the Delta section of Mississippi and Arkansas in 1911 by Norton,[28] from Wisconsin in 1911 by Halloran,[29] and from New York in 1916 by Udall and Fitch.[30] Mohler[31] in 1908 also reported that the disease was more or less prevalent in Kansas, Nebraska, Colorado, Wyoming, Montana, North Dakota, and Texas.

For a period of about 15 years following the recognition of the disease in the United States, it received a considerable amount of attention from the veterinary profession and occupied a prominent place in veterinary literature. During this time, the disease was more or less prevalent in Texas, Nevada, Wyoming, North Dakota, and Minnesota. Each of these states studied the disease at their respective state experiment stations, and extensive studies were likewise made by investigators in the United States Bureau of Animal Industry. However, from 1916 to about 1928 the prevalence of the disease in the United States appears to have decreased, only a few outbreaks being reported during that period.

The gradual disappearance of the disease in Wyoming was observed by French[32] in 1916, who reported that the disease which formerly existed extensively over the state was becoming less general each year.

Scott[18] in 1919 reported that the disease still existed in Wyoming and that it had been definitely reported at different times from at least 14 of the 21 countries in the state but was becoming less prevalent.

Mack[33] from Nevada in 1909 and Van Es[34] from North Dakota in 1911 also reported that losses from the disease were on the decrease.

Wright[35] in 1925 reported that the disease still existed in Nevada but that from 1909 to 1918 was apparently quiescent and caused very little loss.

Kelser[36] reported a severe outbreak of infectious anemia that occurred in 1921 in brood mares of the Remount Depot at Fort Robinson located in northwestern Nebraska. The infection appeared to have been introduced by the purchase of mares from farms in the vicinity of the fort.

Martin[37] reported an outbreak in August of 1928 in a number of army horses of the R.O.T.C. Chicago University unit. The out-

[18] Scott, J. W., 1919. Swamp fever in Wyoming, Wyo. Agr. Exp. Sta. Bul. 121, June.

[19] Brimhall, Wesbrook, and Bracken, 1903. Swamp fever in horses, Repts. of Vet. Dept. of Minn. St. Bd. of Health, St. Paul.

[20] Beebe, W. L., 1905. Swamp fever, 2d Ann. Rept. Minn. Live Stock San. Bd., p. 49, St. Paul.

[21] Peters, A. T., 1906. Malarial fever in horses, Neb. Agr. Exp. Sta., Press Bull. No. 22, Lincoln.

[22] Van Es, L., 1907. 17th Ann. Rept. of N. D. Agr. Exp. Sta., p. 48, Fargo.

[23] Francis, M. and Marsteller, R. P., 1908. Infectious anemia of the horse, Tex. Agri. Exp. Sta. Bul. 119, Austin.

[24] Flower, E. P., 1908. Report of La. St. Live Stock San. Bd., Proc. of 13th Ann. Meet. of Interstate Ass'n of Live Stock San. Bds., p. 162, Chicago.

[25] Whitehouse, A. W., 1909. Notes on swamp fever or infectious anemia of horses, The *Ranchman's Reminder*, VI, p. 3, Laramie.

[26] Kinsley, A. T., 1911. Equine iafectious anemia, Proc. of A.V.M.A., p. 118, Philadelphia, 1910. Rept. of 14th Ann. Meet. of U. S. Live Stock San. Ass'n. p. 143, Chicago, 1910, and *Amer. Jour. Vet. Med.* VI, p. 378, Chicago.

[27] Thatcher, 1910. 20th Ann. Rept. of St. Agr. Exp. Sta. of Wn, p. 13, Pullman.

[28] Norton, O. M., 1911. A practitioner's view of swamp fever, *Amer. Jnl. Vet. Med.*, VI, p. 672, Chicago.

[29] Halloran, D. J., 1911. Infectious anemia, *Amer. Vet. Rev.*, Vol. 38, No. 5, pp. 671-673.

[30] Udall, D. H. and Fitch, C. P., 1915. Preliminary report on the recognition of swamp fever or infectious anemia in New York State, Rept. of N. Y. St. Vet. Col. at Cornell Univ. for the year 1914-1915, pp. 50-62.

[31] Mohler, J. R., 1908. Three diseases of animals which have recently assumed importance to the state sanitarian, *Amer. Vet. Rev.*, XXXIV, pp. 198-202.

[32] French, A. W., 1917, 1919. Bien. Repts. of the St. Vet., Wyo., 1916-1918, Cheyenne.

[33] Mack, W., 1909. Equine anemia, Bul. 68, Nev. Agric. Exp. Sta. 1909.

[34] Van Es, Harris, and Schalk, 1911. Swamp fever in horses, Bul. 94, N. D. Agr. Exp. Sta.

[35] Wright, L. H., 1925. Further investigations of infectious equine anemia in Nevada, U.S.D.A. *Jnl. Agr. Res.*, XXX, Jan.-June 1925, pp. 683-691.

[36] Kelser, R. A., 1922. Equine infectious anemia, *Jnl. A.V.M.A.*, LXII, N.S. 15, pp. 319-331.

[37] Martin, A. D., 1933. Equine infectious anemia, *Vet. Bul. Sup. to The Army Med. Bul. for The Vet. Corps, Med. Dept., U. S. Army*, XXVII, pp. 246-252.

break occurred in horses pastured on a thousand acre tract in the Fox River valley, 50 miles west of Chicago, Illinois. The source of the infection in this outbreak was not definitely determined.

About 1928 reports of outbreaks of the disease in the Mississippi Delta and other sections were received by the Bureau, and in order to obtain information on the prevalence and distribution of swamp fever in the United States at the time, inquiries were made of each state veterinarian. The replies indicated that swamp fever existed during the five years previous to 1929 in the following 17 states: Arkansas, Colorado, Florida, Idaho, Kansas, Louisiana, Michigan, Mississippi, Montana, Nebraska, Nevada, Oregon, Tennessee, Texas, Utah, Wisconsin, and Wyoming. In the states of Colorado, Florida, Kansas, Louisiana, Michigan, Montana, Nebraska, Nevada, Oregon, Tennessee, Texas, Utah, Wisconsin, and Wyoming, the disease was reported to be on the decrease and of little or no economic importance, while in Mississippi, Arkansas, and Idaho the disease was reported to be on the increase and was considered to be an economic problem. Swamp fever was reported to be especially prevalent in the Delta region of Mississippi, where it constituted a major economic problem.

Outbreaks Reported in Recent Years

Although the prevalence of the disease appeared to be on the wane during the period from 1916 to 1928, reports of sporadic outbreaks occurring in various parts of the United States subsequent to that time and studies recently carried on by the Bureau on its distribution indicate that it is again becoming more prevalent.

During the last decade (1930 to 1940) the existence of the disease has been reported from the following states: from Oklahoma in 1930 by Nickel[38]; from Indiana in 1933, where diagnosis was confirmed by horse inoculation test, by the Agriculture Experiment Station, Purdue University[39]; from Montana in 1934 to 1940, where the disease exists in two defined districts and where diagnosis was confirmed by horse inoculation tests, by the Montana Livestock Sanitary Board[40, 41, 42, 43]; from Kansas in 1933 and 1934 by Mercer;[44] from Oregon in 1935 and 1936 by White;[45] from New York in 1933-1934 by Griffin and Brose,[46] who confirmed the diagnosis by horse inoculation test; from Massachusetts in 1936 by Kelser[47] (this outbreak occurred in horses of the R.O.T.C. unit at Harvard University and appears to be the first recorded occurrence of the disease in the New England states); from Vermont in 1939 and 1940 by Jones;[48] from Mississippi in 1939 and 1940 by Gates;[49] from Louisiana in 1940 by Goodwin.[50] Reports of outbreaks of the disease were also made to the Bureau from New York, Michigan, Idaho, Washington, Nebraska, Colorado, Wyoming, and Texas.

In studies on the distribution of the disease during the period from 1935 to 1940, investigators in the Bureau of Animal Industry have recovered the virus from field cases in New York, Maryland, Virginia, Mississippi, Louisiana, Pennsylvania, California, Texas, and Wyoming.

In recent years field investigations of the disease were made by the Bureau, and virus recovered from suspected cases in the East Shore of Maryland, Northern Virginia, the Delta Section of Mississippi, and in the Jackson Hole section of Wyoming, along the valley of the Snake River in the vicinity of the towns of Jackson and Wilson. It is worthy of mention that all these sections, with the possible exception of portions of Virginia, are low-lying, poorly-drained

[38] Nickel, C. C., 1930. Infectious anemia, *No. Amer. Vet.*, XI, pp. 23-24.

[39] Infectious anemia or swamp fever in horses, Ind. Agr. Exp. Sta., Rept. of the Dir. for the Year Ending June 30, 1933, p. 53.

[40] Swamp Fever, Rept. Mont. Livestock San. Bd., Dec. 1, 1934 to Nov. 30, 1936, p. 9.

[41] Swamp Fever Rept. Mont. Livestock San. Bd., Dec. 1, 1936 to Nov. 30, 1937, p. 7.

[42] Swamp Fever, Rept. Mont. Livestock San. Bd., Dec. 1, 1936 to Nov. 30, 1938, p. 4.

[43] Rept. Mont. Livestock San. Bd., Dec. 1, 1939 to Nov. 30, 1940.

[44] Mercer, J. H., 1934. Horse pest in southeastern Kansas, 15th Bien. Rept. of Kan. Livestock San. Commr., 1933-1934, pp. 104-108.

[45] White, S. T., 1936. Infectious anemia, *Rept. Dept. Agr.*, Oreg., April 1, 1935 to Dec. 31, 1936, p. 31.

[46] Griffin, C. A. and Brose, C. P., 1936. Report of an outbreak of equine infectious anemia, with observations on blood changes, *Jnl. A.V.M.A.*, LXXXIX, N.S. XLII, pp. 664-670.

[47] Kelser, R. A., 1936. Equine infectious anemia in New England, *Vet. Bul. Sup. to the Army Med. Bul. for the Vet. Corps, Med. Dept., U. S. Army*, 1940, XXX, pp. 193-196.

[48] Jones, E. H., 1940. 20th Bien. Rept. Commr. of Agr., Ver., 1939, 1940, p. 98.

[49] Gates, W. L., 1940. Equine infectious anemia (swamp fever), *Jnl. A.V.M.A.*, Vol. XCVI. No. 755, pp. 195-199.

[50] Goodwin, J. A., 1940. Mule practice in the sugar cane belt, *Jnl. A.V.M.A.*, XCVI, p. 596.

areas, containing considerable swampy ground where various types of flying, biting insects are numerous during the summer season.

Discussion

In reviewing the history of the disease in the United States, it is seen that subsequent to its definite recognition in 1903 and up to and including 1940 it has been authentically reported from at least 29 of the 48 states, namely, Arkansas, California, Colorado, Florida, Idaho, Illinois, Indiana, Kansas, Louisiana, Maryland, Massachusetts, Michigan, Mississippi, Montana, Nebraska, Nevada, New York, North Dakota, Oklahoma, Oregon, Pennsylvania, Tennessee, Texas, Utah, Vermont, Virginia, Washington, Wisconsin, and Wyoming.

In 15 of these states, diagnosis was confirmed by horse inoculation tests, while diagnosis in the other 14 states was based upon clinical diagnosis. In view of the difficulty of diagnosis in chronic and latent forms of the disease, it is highly probable that infection exists unrecognized in other areas and is more widespread than reports indicate.

Considering the history associated with various reported outbreaks, field investigations made of the disease in different areas, and observations made on the disease under experimental conditions, there appears to be considerable evidence to indicate that following an outbreak involving a large number of horses the most susceptible animals acquire the disease in an acute, subacute, or chronic form and die off, while the more resistant animals appear to recover but remain carriers of the disease. The disease which seems to have waned may thus still exist in a quiescent form for long periods of time without being recognized.

Since experimental evidence indicates that the offspring of infected mares may harbor the virus, the infection may be passed on from generation to generation and thus may be perpetuated indefinitely by hereditary transmission.

The recurrence of outbreaks in certain well defined localities over a long period of years would seem to be more logically explained by the presence of carriers and their infected offspring rather than the possible existence of other sources of virus in nature in those areas.

New outbreaks in formerly infected territory may occur as the result of flare-ups in carrier cases or the introduction of susceptible horses into infected territory and their subsequent exposure to recovered carriers. Outbreaks in uninfected territory usually follow the introduction of infected horses into new territory where conditions are favorable for transmission to susceptible horses.

Summary

Infectious anemia, which was reported from Europe as early as 1843, was first recognized as a specific disease of horses in North America in the Province of Manitoba, Canada, about 1880. Although evidence points to the fact that the disease had existed for at least 50 years in the United States, it was not definitely diagnosed as a specific disease of horses until first recognized in Minnesota in 1903.

During the period from 1903 to 1940 it has been authentically reported from 29 of the 48 states. In 15 of these states, diagnosis was confirmed by horse inoculation test.

Although numerous outbreaks of the disease were reported from widely scattered areas during the period from 1903 to 1916, its prevalence from 1916 to about 1928 appeared to be on the wane. However, during the last decade (1930 to 1940) the existence of the disease has been reported from a number of new areas and again appears to be increasing in prevalence.

Since 1935, investigators in the Bureau of Animal Industry have recovered the virus from suspected field cases in New York, Maryland, Virginia, Mississippi, Louisiana, Pennsylvania, California, Texas, and Wyoming.

With the exception of the Mississippi Delta where the disease appears to be well established and exists principally in the chronic form, outbreaks are chiefly of a sporadic nature, being confined to small areas and showing little or no tendency to spread.

The Microscopic Detection of Bacterial Defects of Milk*

By C. S. BRYAN, East Lansing, Michigan
Division of Veterinary Science, Michigan State College

Fig. 1. Insanitary conditions on the farm promote disease among animals and result in a milk of very low quality with a high bacteria count

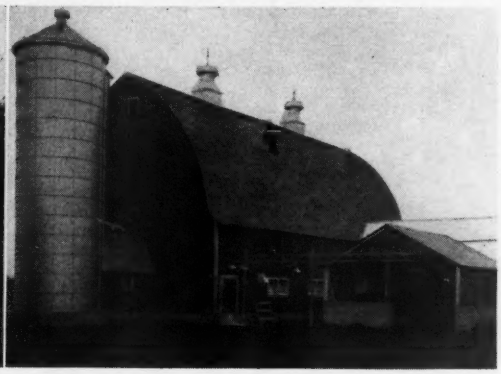


Fig. 2. Well-planned buildings of this type, with concrete manure pit and properly drained yard, aid in quality milk production.

THE cows that produce market milk must be healthy, especially free from udder infection, clean, and stabled in sanitary quarters if the milk produced is to be safe and of reasonably high quality. The equipment used to handle this milk must be of proper construction, clean, and handled in a sanitary manner if the quality of milk is to be maintained.

The presence of infectious mastitis in the lactating cows and the lack of application of proper and adequate sanitary procedures in handling the milk contribute bacterial contamination to the milk. These bacteria can be counted and the types noted upon microscopic examination of appropriate milk samples. The detailed procedures for doing this follow.

The Microscopic Test for Infectious Mastitis

Milk samples may be collected at any time except within two hours after the regular milking. The following are the steps taken in collecting the milk:

1. Wash udders and teats with chlorine solution (200 p.p.m.).
2. Discard two streams from each quarter into a strip cup.
3. Collect a 5cc cow composite milk sample by milking directly into the sterile tube containing the differential preservative, being careful not to touch the part of the cork that goes into the tube.
4. Mix by inverting the tube several times.
5. Identify samples properly, using indelible or wax pencil on cork.
6. Take or mail to laboratory.

The differential preservative mentioned in step 3 is not essential, even though desirable, if the test is made within 24 hours of the collection of samples. It is prepared by adding 0.75gm sodium azide, 0.2gm brilliant green, and 10gm dextrose to 200cc distilled water. This is filtered, or allowed to settle, the supernatant portion being used, and sterilized at 15 pounds pressure for 20 minutes. It is dispensed aseptically at the rate of 0.1cc per sterile tube for 5cc of milk. The final dilutions of the chemicals in the milk are: sodium azide, 1-15,000; brilliant green, 1-50,000; dextrose, 1-1000.

The milk samples are incubated at 37°C. (98.6°F.) for 12 to 15 hours, and slides for microscopic examination are then prepared. To prepare the slides, use a loop of 4mm outside diameter (this can be made by bending 24-gauge wire around a 6 d. wire nail). Spread one loopful of well-mixed milk over an area of 4x8mm, placing 20

*Presented at the Michigan State College Post-Graduate Conference for Veterinarians, East Lansing, Mich., January 27-February 1, 1941.

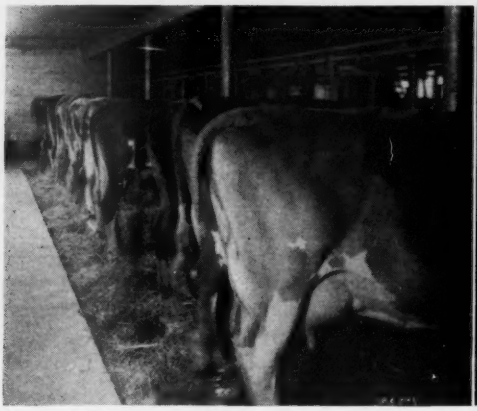
smears on each slide. Identify the slide, using a wax pencil. After the film has been allowed to air dry, it is fixed by heating in flame until just too hot to touch and cooling, repeating this process about three times. The slide is then ready for staining.

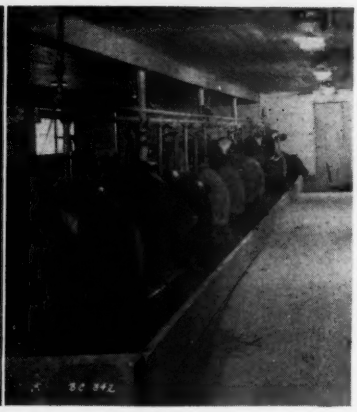
To stain the slides, place them in the following solutions in order, for about one minute, draining between solutions: (1) xylol, (2) 95% alcohol (or 70% rubbing alcohol), (3) methylene blue stain. The methylene blue stain is prepared by adding 10cc of saturated alcoholic solution of methylene blue to 90cc of 30% alcohol. (To make 30% alcohol, add 30cc of 95% alcohol to 65cc of water, or 30cc of 70% rubbing alcohol to 40cc of water.)

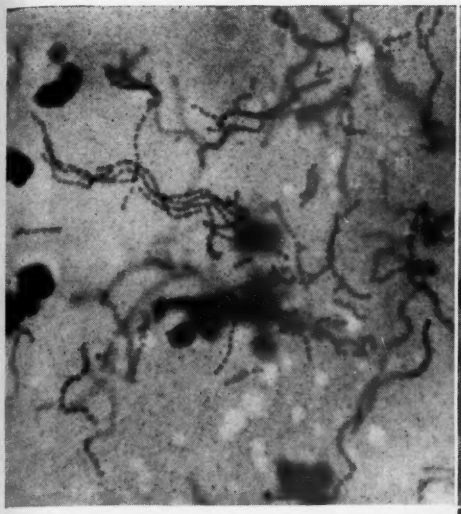
Alternative staining solutions are Newmann's or Broadhurst's single dip stain. Newmann's stain consists of 1 to 1.2gm methylene blue, 65cc of 95% alcohol, 40cc of tetrachlorethane, and 6cc of glacial acetic acid. When this is used, the slides should be dipped in the solution, then dried before using water to wash off the excess stain.

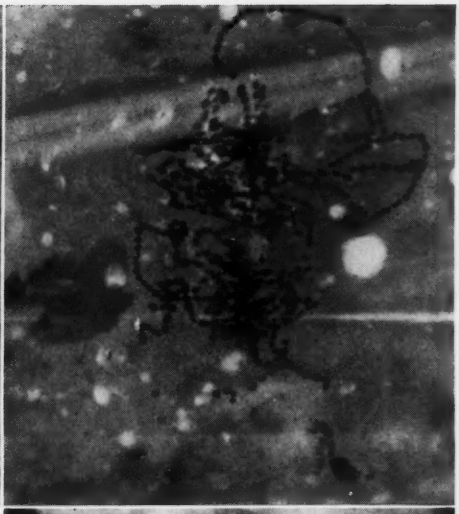
Broadhurst's stain is made as follows: To 54cc of 95% alcohol add 0.4cc of concentrated sulphuric acid c.p. Mix with 40cc technical tetrachlorethane in flask and heat to 55° C. (no higher). Add while hot 1 to 1.2gm of methylene blue dye, and shake to dissolve dye. Then add 8cc of 1% alcoholic solution of fuchsin (1gr basic fuchsin), and

Figs. 3, 4. Inadequate equipment results in poor cooling and adds to the hazards of production
Figs. 5, 6. A clean barn and well-kept herd are the first requisites in producing high-quality milk









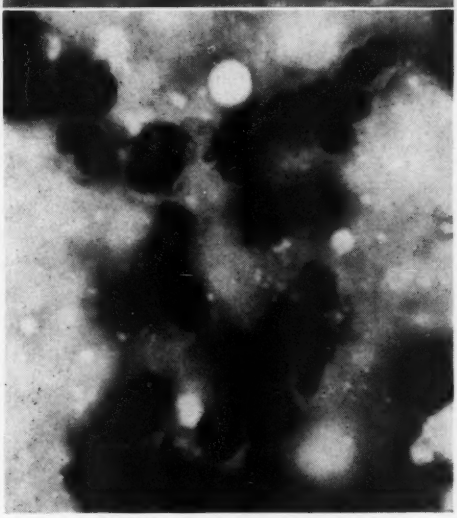


Fig. 7. Infected: Streptococci and leucocytes in excess of 1 million per cc. X 1500

Fig. 8. Infected: Less than 1 million leucocytes per cc, but streptococci present. X 1500

Fig. 9. Suspected: No streptococci, but leucocytes in excess of 1 million per cc (retest within one month). X 1500

keep in tightly stoppered bottles. To use, dip the slide into the stain or flood for 15 seconds, drain and dry; wash in cold water till all blue is washed out and smear becomes pink. The bacteria and leucocytes will then be blue and the background faint pink.

After staining, the slides are read, using an oil immersion lens and 6x ocular. The cow is *infected* when streptococci (more than six cocci per chain) are present, with or without an increase in the number of leucocytes. (Leucocytes up to and including 1,000,000 per cc are considered normal; that is, up to and including three per field, if the factor is 300,000.) The cow is a *suspect* and should be retested within a month when an increased number of leucocytes is present with no streptococci. The cow is *negative* with less than 1,000,000 leucocytes and no streptococci in her milk. Staphylococcic or *Escherichia coli* mastitis is suspected when either of these bacteria appears to be present in the milk together with an increase in leucocyte count (above 1,000,000) in the absence of streptococci, if there is a history of acute mastitis.

The Direct Microscopic Count and Interpretation

Samples are collected with a dipper directly from the cans or weigh vat. The dipper is rinsed in running water between samples, or rinsed thoroughly in the next milk before sampling. The samples are placed in sterile tubes and iced. (If the slides are prepared in the plant, use the loop to collect the samples.)

The slides are prepared in the same manner as in the test for mastitis, and the oil immersion lens, and 6x ocular are used.

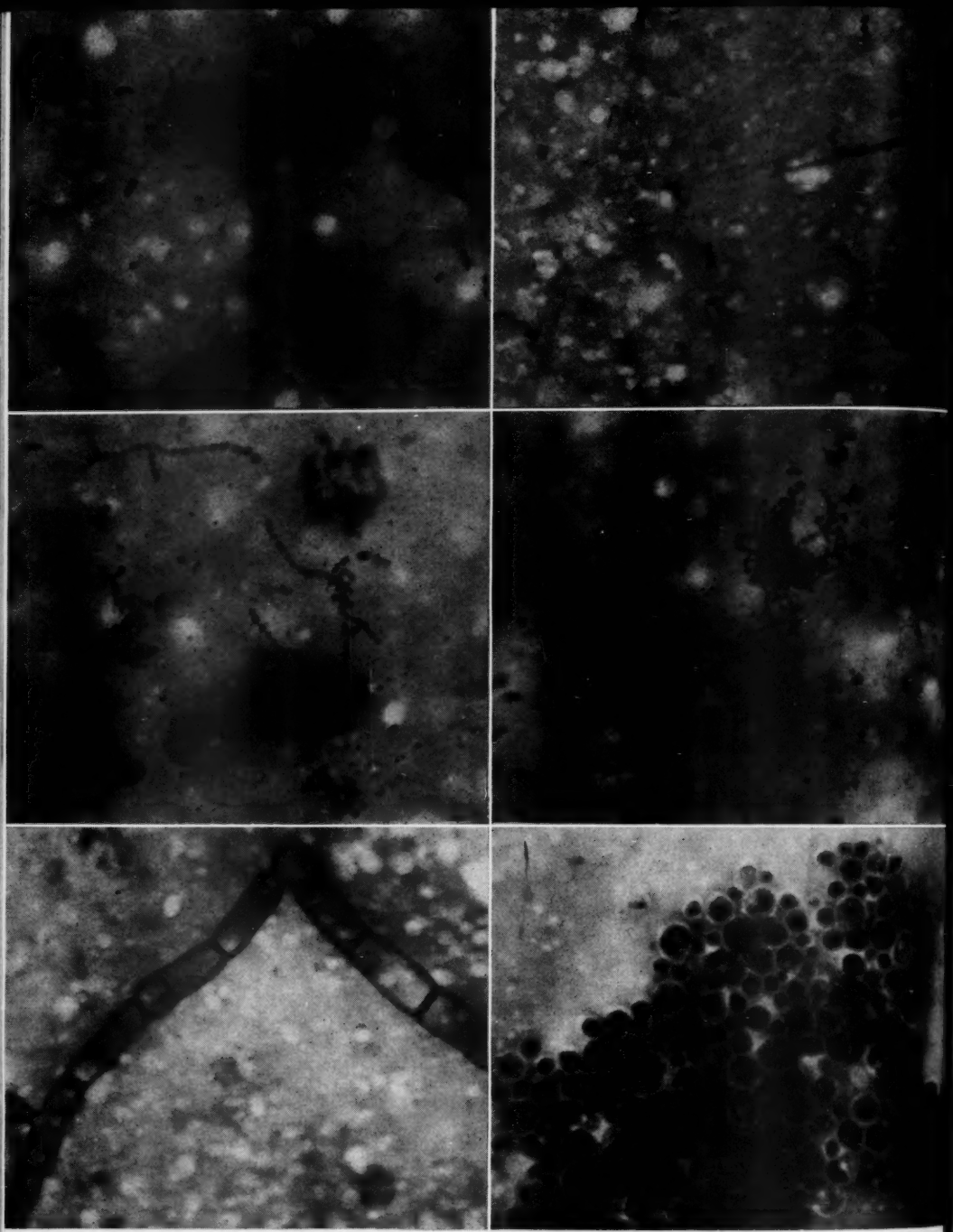


Fig. 10. The microscopic appearance of milk exposed to excess contamination from dirty cows or barn (large rod bacteria) and dirty utensils, short, paired rods). In addition, an increased number of cells are present in the milk. X 1500

Fig. 11. The normal milk-souring bacteria are desirable in milk unless they are present in large numbers. Their action of souring milk should be delayed by proper cooling, thereby increasing the keeping quality of the milk. The presence of *Str. lactis* in large numbers indicates poor cooling. X 1500

Fig. 12. The microscopic appearance of milk from a herd with *Str. mastitis* infection, as indicated by the streptococci and cells. The milk was handled in dirty utensils, as indicated by the cocci in singles and clumps and the short, paired rods. X 1500

Fig. 13. The microscopic appearance of milk produced in a dairy insanitary in every respect, as indicated by many bacteria of various types. X 1500

Fig. 14. Mold mycelium or spores in milk indicate dust contamination. Dust in the barn or milkhouse is the source of this contamination. X 1500

Fig. 15. When milk utensils are exposed to dust contamination in the milkhouse, yeast cells are usually present. The yeast cells find suitable lodgment in unclean dairy utensils. X 1500

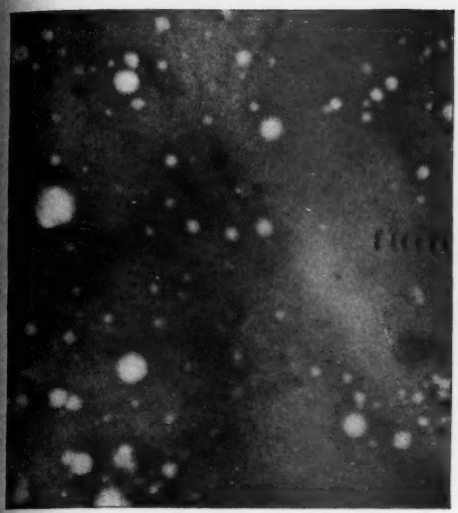


Fig. 16. The microscopic appearance of high-grade milk. Few, if any, bacteria are found in milk produced and handled under sanitary conditions. X 1500

If casual observation indicates a high bacteria count, count 10 fields and average to get the average number of bacteria per field; if a low count is indicated, check 50 fields. The bacteria count is obtained by multiplying the average number of bacteria per field by the proper factor. The present-day microscopes are built for milk work with the diameter of the microscopic field set at 0.205mm, yielding a factor of 300,000. The number of bacteria per cc is therefore 300,000 times the average number of bacteria per field.

The following is a handy table for use in making microscopic counts:

Bacteria in fields			=	*bacteria count*
1	"	50	=	6,000
1	"	25	=	12,000
1	"	12	=	25,000
1	"	6	=	50,000
1	"	4	=	75,000
1	"	3	=	100,000
1	"	2	=	150,000
1	"	1	=	300,000
2	"	1	=	600,000
3	"	1	=	900,000
4	"	1	=	1,200,000
5	"	1	=	1,500,000
6	"	1	=	1,800,000
7	"	1	=	2,100,000
8	"	1	=	2,400,000
9	"	1	=	2,700,000
10	"	1	=	3,000,000

If clumps or singles are counted as one, the microscopic count will be about the same as the old standard plate count (using plain nutrient agar); if all individual bacteria are counted, the count will be about the same as the standard plate count (using tryptone glucose extract medium).

The suspected source of excess contamination on the farm is indicated only where the bacteria count exceeds 100,000 per cc of milk. The following table shows the probable causes of various types of contamination:

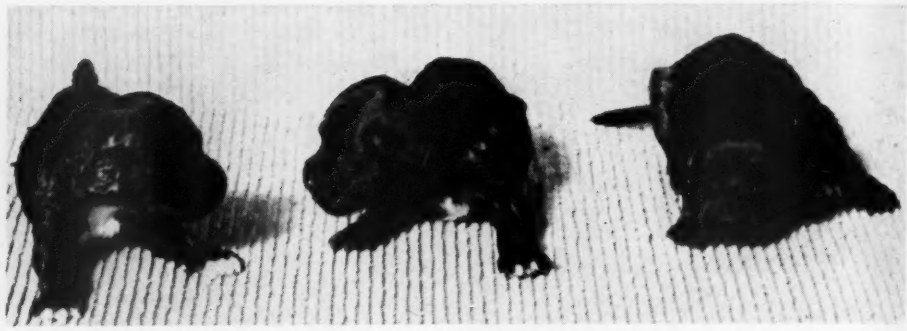
	Shape and Type of Organisms	*Probable Cause*
Rods	Short paired (Scattered, clumps or in chains)	Utensils—wet, unclean surfaces, especially milking machines
Rods	Short, thick Long, thick (Scattered, clumps or in chains) Long, thin Short, thin	Dirty cows or barn—manure, dust, dirt, wet milking, flanks
	Very high counts (with high temperature)	Poor cooling
Cocci	Short chains (2-5 elements, *Str. lactis*	Utensils—scum accumulations in crevices or open seams
Cocci	Clumps or singles (staphylococci)	
Cocci	Tetrads (group of 4) (scattered or clumps)	Dirty cows or barn
Cocci	Streptococci (more than 6 elements)	*Str. mastitis*
	Various types scattered throughout (insanitary in every respect)	Poor production
Mold	(spores or mycelium)	Dust—in barn or milk-house
Yeast	(especially when milking machines are kept in dusty atmosphere)	Dust—in barn or milk-house
Cells	Polymorphonuclear Lymphocyte Epithelial	One of the following: 1. Milk used too soon after freshening 2. Milk used too long at end of lactation period. 3. Injury to udder (traumatic) 4. Streptococcic mastitis if streptococci are found in unincubated or incubated milk samples.

Summary

The microscope is essential in a quality milk control program to determine the presence or absence of infectious mastitis in the cow, to determine the bacterial count of milk and, in the case of high counts, to indicate the probable source of excess contamination as revealed by the types of microorganisms present in poor quality, high count milk.

Vaccine Dosage in Canine Distemper

By LEON F. WHITNEY,[1] Orange, Connecticut



SINCE the publication of the author's previous paper on this subject, "Permanent Distemper Immunization by Vaccine Alone,"[2] an additional number of puppies have been vaccinated in his kennels by this method, with some interesting results. In that paper, one of the points brought out by the data was the need for fresh vaccine, and another was the advisability of grading the dose to the size of the dog instead of giving all dogs, large and small, the same dose of 5cc, as is the present custom.

The developments from two new groups of puppies may now be added to the previous data, and the whole body of data examined critically. The new data cover dogs vaccinated with six-day-old vaccine—the same age as the vaccine used previously. It was made in Auburn, Alabama, and shipped by parcel post to New Haven, Connecticut.

The first group vaccinated consisted of 11 cocker spaniels weighing four to eight pounds each. Each was given two doses of vaccine. The first dose was 2.5cc; the second 3cc, given two weeks later.

The second group (48 puppies) received a single dose of 5cc, one month after the first group received its first dose. No virus was administered in either group. About four months after the first and three months after the second group was vaccinated, a dog sick of distemper was introduced into the kennels. The disease spread gradually through the kennels. Twenty-eight of the 48 animals in Group II, and all of a control group of seven, contracted the disease. Some were very sick. This is shown in Table I, in which the severity of the attack is indicated by the number of X's; that is, as well as the severity of the illness could be judged by the general picture of the disease: prostration, anorexia, temperature, loose, discolored feces, etc. The weight of each puppy at the time of vaccination is also recorded in Table I. By the time they became infected they had all grown larger and some were full grown.

Of the 48 in Group II (Table I), 20—the smallest ones—were solidly immune. Twenty-eight contracted the disease, of which 26 lived and two died. Table I shows that the approximate severity of the attack was closely correlated with the weight at the time of vaccination; that is, with the amount of vaccine per pound of body weight. None of the 11 little puppies included in Group I contracted the disease, and hence are regarded as being solidly immune.

Of the seven unvaccinated controls, all contracted distemper. Two of these controls were bullhounds, almost full grown. Both recovered. Four were cocker spaniels; two lived and two died. The remaining control, a bloodhound puppy, died.

[1] The author wishes to express his gratitude to the White Isle Kennel Manager, Mr. Edward Lacourciere, and to Mrs. Alice Demers, for their careful and painstaking work in caring for the dogs throughout this experiment.

[2] *Vet. Med.* 35:7, pp. 408-414.

TABLE I.—CORRELATION OF WEIGHT OF PUPPY AT TIME OF SINGLE 5CC VACCINATION, WITH SEVERITY OF DISTEMPER SYMPTOMS.

No.	Breed	Weight	Severity
1	Bloodhound	54	XXXX
2	Bullhound	43	XXX
3	Bloodhound	42	XXX
4	Bloodhound	41	XXX
5	Bloodhound	40	XXX
6	Bullhound	40	XX
7	Bloodhound	28	XXX
8	Bloodhound	27	XXX
9	Bloodhound	25	XXX
10	Bloodhound	22	XXXX
11	Bloodhound	21	XX
12	Bloodhound	20	XXX
13	Bloodhound	20	XXX
14	Bloodhound	19	Died
15	Bloodhound	19	XX
16	Bloodhound	17	XXX
17	Bloodhound	16	XXX
18	Bloodhound	16	Died
19	Bloodhound	16	XX
20	Bloodhound	16	XX
21	Bloodhound	15	XX
22	Bloodhound	15	X
23	Bloodhound	14	XX
24	Bloodhound	13	X
25	Bloodhound	13	XXX
26	Bloodhound	13	X
27	Bloodhound	12	X
28	Bloodhound	12	X
29	Bloodhound	10	.....
30	Cocker Spaniel	10	.....
31	Cocker Spaniel	9	.....
32	Cocker Spaniel	9	.....
33	Cocker Spaniel	9	.....
34	Cocker Spaniel	9	.....
35	Cocker Spaniel	8	.....
36	Cocker Spaniel	8	.....
37	Cocker Spaniel	8	.....
38	Cocker Spaniel	8	.....
39	Cocker Spaniel	8	.....
40	Cocker Spaniel	7	.....
41	Cocker Spaniel	7	.....
42	Cocker Spaniel	7	.....
43	Cocker Spaniel	7	.....
44	Cocker Spaniel	7	.....
45	Cocker Spaniel	6	.....
46	Cocker Spaniel	6	.....
47	Cocker Spaniel	5	.....
48	Cocker Spaniel	5	.....
	CONTROLS.		
1	Bullhound	42	XXX
2	Bullhound	40	XXX
3	Bloodhound	67	Died
4	Cocker Spaniel	21	XXXX
5	Cocker Spaniel	15	Died
6	Cocker Spaniel	15	Died
7	Cocker Spaniel	14	XXXX

The outbreak of distemper introduced via a very sick dog did not extend beyond the two groups of puppies, although the remainder of the kennel was equally exposed. All the other dogs, many of which were there during the study last reported, were solidly immune. Some of the dogs were sick for many weeks. Almost three months elapsed between the beginning of the attack and the recovery of the last dog. When the epizootic was over, a dog almost dead with distemper was sacrificed and its spleen was used to make a 20% suspension in saline solution. Twenty cc of this was injected into one of the recovered dogs, and a second 20cc the following day. Several abscesses occurred, but the dog showed no other effects.

Incidentally, a number of the dogs in this study have been exposed to "canine influenza," which appeared in the kennels in January, 1941, and none have contracted it, although none had been exposed previously. This adds further weight to the author's opinion that dogs which have had distemper are immune to "canine influenza."[3]

Three puppies, vaccinated at Auburn, gave a further illustration of the lessened value of stale vaccine. These were about eight weeks old. They were given two doses of commercial vaccine, two weeks apart, and a dose of virus two weeks later. This was procured from the local dealer. He had obtained it from the state distributor, who in turn had obtained it from the manufacturer, 1100 miles away. The puppies were sent home and exposed. They all lived, but were among the sickest puppies in the kennels. (They are not included in the tables.)

Some difficulty was experienced in making sterile vaccine.[4] As it became older, it did not remain sterile, despite the 0.2% of formalin used to attenuate the virus. A small batch of vaccine containing 0.4% of formalin was therefore made. This was injected into four cocker spaniel puppies when it was 48 hours old. It solidly immunized these dogs, as demonstrated by subsequent exposure, but proved to be impractical because of the irritation it caused at the site of injection. The puppies cried, scratched and chewed at the injected sites for some time. The vaccine remained sterile during the two weeks it was kept.

Combining these data with those recorded previously,[2] let us examine them for any further light they may shed on the following problems: Is fresh vaccine much superior to stale? Is the usual dose now given

[3] Whitney, Leon F., 1941. "Canine influenza." *Vet. Med.* 36:4, pp. 216-219.

[4] Making distemper vaccine free from all bacteria, even when formalized with 0.2% formalin, as suggested by Laidlaw and Dunkin, is a difficult procedure. As Pyle has shown, most spleens are naturally infected with various organisms, so it is likely there will be some proliferation unless preservatives are added. Because of this, the commercial producer is able to put out a product far safer than that which the veterinarian can produce with the equipment he has available. The author has encountered many more abscesses when using his own vaccine than he has when using commercial material.

TABLE II.—DOGS RECEIVING ONE OR TWO 5CC DOSES OF VACCINE.

No.	Breed	Weight	Amt. Vac.	Distemper: Immune	Distemper: Recovered	Distemper: Died
16	Bloodhound	74	5-5		X	
1	Bloodhound	70	5-5		X	
3	Bloodhound	70	5-5			X
2	Bloodhound	68	5-5		X	
23	Greyhound	56	5-5	X		
26	Greyhound	55	5-5	X		
19	Bullhound	55	5-5		X	
4	Bloodhound	52	5-5		X	
5	Bloodhound	52	5-5			X
22	Bullhound	48	5			X
25	Greyhound	35	5	X		
33	Beagle	23	5-5	X		
31	Beagle	26	5-5	X		
30	Beagle	22	5	X		
32	Beagle	21	5	X		
6	Bloodhound	20	5			X
28	Cocker spaniel	15	5-5	X		
36	Cocker spaniel	13	5-5	X		
37	Cocker spaniel	11	5-5	X		
19				10	5	4

TABLE III.—DOGS RECEIVING TWO DOSES OF 2.5CC OF VACCINE TWO WEEKS APART.

No.	Breed	Weight	Distemper: Immune	Distemper: Recovered	Distemper: Died
20	Bullhound	51		X	
12	Hound	34	X		
9	Hound	22	X		
8	Bloodhound	21			X
13	Bloodhound	20	X		
7	Bloodhound	18			X
29	Scottie	17	X		
34	Cocker Spaniel	14		X	
35	Cocker Spaniel	12		X	
38	Cocker Spaniel	11		X	
39	Cocker Spaniel	11		X	
40	Cocker Spaniel	11		X	
41	Cocker Spaniel	11	X		
13			5	6	2

adequate? Is one dose equally effective as two smaller doses?

After some rearrangement of the previous data for the sake of convenience, the following groups can be distinguished: One group (Table II) received two doses of 5cc each of vaccine, followed by a dose of virus. (Since almost no reactions were observed after the virus was given, it would seem that we are warranted in concluding that the virus was dead for the most part, so that this lot of dogs is comparable to those in which no virus was used.) A second group (Table III) resembled the first in every respect except that the dose of vaccine was only 2.5cc (administered twice). A third group (Table I) was given a single dose of 5cc of vaccine; a fourth (Table IV), two doses of 5cc; a fifth (Table V) one dose of 5cc or 3cc; and a sixth (the group of 11 cocker spaniels described above), two doses of 2.5 and 3cc, respectively.

The most obvious fact that appears in studying these tables is that there is considerable variation in the potency of the vaccines used. Whether this variation results from aging, from having been exposed to heat while in the mail, from improper storage, or from other causes, it is impossible to say. At any rate, it seems advisable to use a goodly amount of vaccine at all times, to offset the possibility of lower potencies in some batches.

Another fact which stands out is that vaccination is decidedly worthwhile, whatever method is used. Of the 169 dogs vaccinated by all the methods, 86 were solidly immunized, 59 contracted distemper but recovered, and only 24 died. Of the 155 controls, one was immune, 47 contracted distemper and lived, and 107 died.

Again it became obvious that among the larger dogs no group was as adequately protected as that which received 48-hour-old vaccine (Table V). Apparently, the fresher the vaccine, the better.

TABLE IV.—DOUBLE VACCINATION, TWO-WEEK INTERVAL.

Nos.	Breed	Appr. Weight (pounds)	Amt. Vaccine (in cc)	Distemper: Immune	Distemper: Recovered	Distemper: Died
1	Bloodhound	75	10 and 5	0	1	0
2	Dane x bloodhound	70	10 and 5	0	0	1
3– 4	Bloodhounds	50	5 and 5	0	0	2
5–10	Bloodhounds	25	5 and 5	0	3	3
11–13	Foxhounds	16	5 and 5	0	2	1
14–23	Cockers	15	5 and 5	3	5	2
24–26	Scotties	11	5 and 5	3	0	0
27–35	Cockers	11	5 and 5	2	4	3
36–41	Cockers	5	3 and 3	3	2	1
41				11	17	13

TABLE V.—SINGLE VACCINATION WITH FRESH (48-HOUR) VACCINE

Nos.	Breed	Appr. Weight (pounds)	Amt. Vaccine (in cc)	Distemper: Immune	Distemper: Recovered	Distemper: Died
1– 5	Bloodhounds	55	5	2	3	0
6– 8	Greyhounds	50	5	3	0	0
9–11	Bloodhounds	30	5	2	1	0
12–16	Bullhounds	15	3	5	0	0
17	Cocker	22	3	1	0	0
18–20	Beagles	9	3	3	0	0
21–24	Cockers	12	3	4	0	0
25–31	Cockers	8	3	7	0	0
31				27	4	0

Each of the tables shows that when approximately the same size dose was given to all the dogs, the smaller animals were better protected. This is eloquent testimony that 5cc or even two doses of 5cc each, are not enough to protect large dogs solidly against distemper. Table I shows this best of all. With a few exceptions, the severity of the disease varied with the size of the dog.

Lastly, the data show that sometimes one dose is as effective as two; and that one dose of *fresh* vaccine is decidedly more effective than two doses of *stale*. Comparing the data in Table I with those in Table IV, it appears that, even using vaccine of the same age and made under very similar conditions, one dose was actually superior to two. This could scarcely be so; the results must be explained on the basis of variation in the potency of the lots of vaccine.

It appears that the reasoning which prompted the giving of two small doses of vaccine several weeks apart instead of one large dose, was faulty. Does not our knowledge of immunity suggest that it is better to give large doses of antigens to shock the body into proliferation of a large number of antibodies, than to give several small doses? If the body is induced to proliferate a given number of antibodies, and then another dose of antigen of equal size is injected, will not the antibodies already present neutralize at least part of it, so that fewer antibodies are produced by the second dose? If the entire amount were injected the first time, when there are no specific antibodies present, it is reasonable to expect that a much larger number of antibodies would be produced.

In the case of hyperimmunity, where the object is to induce heavy antibody proliferation, it is not the practice to give many small doses over an extended period, but rather to give two huge doses of antigen a day apart. In the immunization of rabbits against washed sheep cells, as conducted in bacteriology laboratories, the animals are no longer given many small injections, but rather two massive ones close together, usually one day apart. Laidlaw and Dunkin showed that a dog requires at least five days to build up protective antibodies against distemper, after he has been vaccinated. If two doses are to be given it would seem that they should be given within a short time rather than to wait two weeks for the second dose.

Another consideration favoring the single dose is the demonstrated superiority of fresh vaccine. If the same batch of vaccine be used for two injections, as is now usually done, the first dose is often none too fresh, and the second, two weeks older than the first, is obviously even less potent.

Since he started practice the author has been using the one-injection method, grading the dose of vaccine to the size of the dog. Very small puppies—those weighing under three pounds—have been given only 5cc of commercial vaccine. Care has been exercised to be sure the vaccine was as fresh as possible. Puppies weighing between five and 10 pounds received 10cc and larger dogs much more. One dog weighing 70 pounds received 30cc. In no case was there any evidence of reaction from these large injections. Apparently they have been perfectly safe. Some of these dogs have since been exposed to distemper, and these were solidly immune.

Conclusions

1. To produce solid immunity against distemper, by the use of vaccine without virus, it is necessary to use fresh vaccine; the older the vaccine, the greater the indicated dose.

2. To produce solid immunity it is necessary to grade the dose to the size of the dog. Five to 10cc of ordinary commercial vaccine does not appear to be enough for a large dog. A dose of at least double that amount is suggested.

3. Even small doses of vaccine are decidedly worthwhile as a protective measure, especially if the vaccine be fresh.

4. Of 169 dogs treated by various methods, 86 were solidly immunized, 59 contracted distemper but recovered, and only 24 died. This is in contrast to the controls, of which 107 of 155 dogs died.

5. The use of 0.4% formalin instead of the usual 0.2% for attenuation and preservation of the vaccine caused an objectionable degree of local irritation of the site of injection.

Recent Materia Medica

XII. The Adrenal Hormones

By PROF. VICTOR LEWITUS

History

THE adrenal glands or suprarenal bodies have but recently become objects of intensive research, probably because they went unnoticed by the ancients. This was probably due to their small size and failure to recognize their specific functions. The ancients at first considered them a portion of the perirenal adipose tissue.

Eustachius, in 1563, first described "small glands which were attached to the upper pole of each kidney." From his description, the term "suprarenal" was derived. For nearly 300 years numerous scholars developed various theories as to the functions of the suprarenals, all since proved erroneous.

In 1716 the Academy of Sciences of Bordeaux offered a prize to the one who could determine the functions of these structures, but none of the contributions submitted were considered worthy of the reward.

During the 18th century slight progress was made when Morgagni first observed the existence of accessory adrenal bodies in the human being, though Hartmann had earlier observed such structures in the dog.

With the advent of the 19th century, comparative studies of the glands were initiated. Meckel (1815) noted the anatomical differences in birds and mammals, though he neglected to discover the glands in reptiles. He suggested the relationship of the glands to the reproductive system. Other workers noted adrenal homologues in various species of fishes and amphibia. In 1839, Bergmann first reported the relationship of the adrenal medulla to the nervous system. An important observation was made by Vulpian in 1856. He noticed that the adrenal bodies contained a powerful reducing substance, so that when the gland was cut, the exposed surface when treated with ferric chloride turned bluish-green, or when in contact with strong alkali or halogens turned reddish-brown. Hence the adrenal bodies were termed "chromaffin" bodies.

Genuine progress in the modern study of the adrenals may be ascribed to the work of Thomas Addison who, in 1849, was first definitely to link systemic disease with pathologic changes in the adrenals. Not only did he describe the signs and symptoms as found in humans, but he correctly determined that the diseased state was due to destruction of the adrenal bodies as a result of tuberculosis. Thus tuberculosis of the adrenals is known to this day as Addison's disease.

Anatomic and Physiologic Considerations

Though a great deal of effort has been spent in comparative anatomical studies, much confusion still exists as to the histology of the structures, due to certain practical difficulties. According to Grollman "many descriptions of the microscopic appearance of the cells of the glands are in reality based on artifacts." Despite these difficulties a great deal of information has accumulated regarding their character. The gland consists of two distinct parts which differ in both origin and function. The outer portion or cortex is derived from the mesoderm, whereas the inner portion or medulla comes from the neural ectoderm. The cortical area contains highly vascularized cells which are arranged in palisade columns at right angles to the periphery. The medulla contains polygonal or oval cells rich in nerve fibers of the sympathetic system. Grollman, along with numerous other authorities, considers the glands in early life as composed of the medulla, the cortex, and "the androgenic tissue," the latter under pathologic states causing disorders of the reproductive system. This view of the tripartite nature of the adrenal gland is supported by many well-established facts. Brown-Séquard as well as other physiologists demonstrated that, following total bilateral adrenalectomy, animals die. Oliver and Schaefer, in 1895, were first to show that an extract prepared from the adrenals produces a marked

increase in blood pressure when injected in experimental animals. Thus it was first established that the adrenals elaborated a hormone.

Epinephrine (Adrenalin), Medullary Hormone

The remarkable discovery of Oliver and Schaefer not only stimulated modern endocrine research, but caused feverish efforts to be initiated in the isolation of the active principle which was responsible for the changes observed by them. Abel in this country, and von Fürth at Strassburg obtained highly concentrated preparations of the medullary portion, which had pronounced activity. However, Takamine first isolated the hormone in 1901—an epoch-making discovery, for this was the first hormonal substance to be definitely determined. In 1904, Stolz (and later Dakin and Flacher) synthesized the hormone, so that today both natural and synthetic forms of epinephrine are available.

Epinephrine is also known as adrenalin or suprarenin. It is chemically methylaminoethanolcatechol, having the formula $C_9H_{13}O_3N$. It occurs as a white or cream-colored crystalline substance, soluble in alcohol, and slightly soluble in water (more so in hot water). It is insoluble in chloroform, ether, acetone, or fixed or volatile oils; has a mildly bitter taste, and is feebly alkaline in solutions. It decomposes gradually in air (becomes oxidized), and when heated to 190°C begins to decompose, melting at 205 to 212°C. When solutions decompose they usually change to a brown or pink color. The most popularly used compound, because of its greater solubility, is epinephrine (adrenalin) hydrochloride, used in topical or parenteral solutions. Epinephrine or its hydrochloride is incompatible with all oxidizing agents (oxygen, chlorine, iodine, iron, etc.) and alkalies.

Epinephrine is standardized both physicochemically and by biological assay. The crystals may be determined on the basis of melting point and optical rotation; whereas solutions may be tested for their activity by injecting dilute solutions into anesthetized dogs. Thus the activity of the levorotatory product (from natural sources) may be compared with that of the racemic product (which has been synthesized). Qualitatively, the activities are identical, as evidenced by the requirements of the U.S.P.

In shock or collapse, particularly following anesthesia, slow intravenous injections of epinephrine are recommended for restoring the blood pressure by correcting the reduced peripheral circulation

which does not specify the origin but merely specifies the levo-rotatory product.

Epinephrine acts by virtue of its effects on the autonomic nerve system, stimulating the myoneural junctions of the sympathetic nerve endings. It acts chiefly peripherally, its most conspicuous activity being evident in constriction of the blood vessels. There is consequently a rise in blood pressure and a slowing of the heart, as well as direct stimulation of the myocardium. Following intramuscular or subcutaneous injections, there is dilatation of the bronchioles due to relaxation of the bronchial muscles. Glycosuria also follows injections. Moderate oral doses usually do not have such effects, though sometimes there are exceptions to this finding. Epinephrine is used locally for its vasoconstrictor effects, and also in certain respiratory congestive states. Upon injection, it is known to relieve asthmatic paroxysms. In emergencies such as shock or excessive anesthesia, intravenous administration sometimes affords surprising results. Its use in conjunction with most local anesthetics is quite widespread, since it not only prolongs the anesthesia by retarding the peripheral circulation, but also lessens the toxicity of the anesthetic by retarding absorption into the general circulation. The duration of action of epinephrine is relatively short.

Application.—Epinephrine, chiefly in the

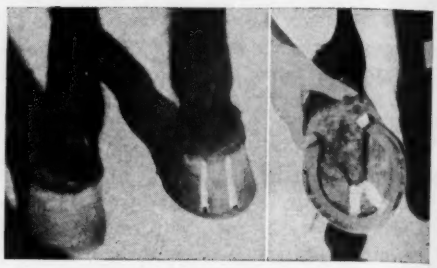
form of the hydrochloride, has but limited use in veterinary medicine. It is used for its vaso-constrictor effects in reducing post-operative hemorrhage, particularly in operations about the eyes or lips. It is frequently used in hypodermic injections of local anesthetics to delay absorption and prolong local anesthesia. It is used also for the temporary relief of congestion of the mucous membranes as in the examination of highly inflamed conjunctivae. In case of shock or collapse, particularly during or following anesthesia, it has often been used heroically and has proven its worth since it restores the blood pressure by correcting the reduced peripheral circulation. Slow, intravenous injections are to be recommended in such cases. Epinephrine is sometimes used by subcutaneous injection in the treatment of laminitis or founder, the drug to be injected in the region of the affected plantar nerve ends. In certain ocular conditions, such as glaucoma, epinephrine is at times used for its myotic properties in 2% solutions of the hydrochloride.

Dosage Forms.—Epinephrine (adrenalin) is usually marketed in a solution of the hydrochloride, 1:1000 for topical or local application (diluted with sterile saline solution up to 1:15,000); for oral inhalation (sprays) there has been made available recently a 1:100 solution; in ointment form (1:1000) for application to mucous membranes of the eyes, nostril, etc.; and in the form of suppositories (1:1000). A suspension of epinephrine in oil in 1:500 strength for intravenous use in humans has recently become available; also a combination of epinephrine with gelatin, called suprarenalin-gelatin mixture. The latter has been introduced for human use (subcutaneously) because of its prolonged effects. It must be gently warmed prior to use.

Dosage.—Epinephrine hydrochloride solution (1:1000) is used subcutaneously as follows: dogs, minims iii to x (0.2 to 0.6cc); cattle and horses, ʒ to ʒii (4 to 8cc). For intravenous use, the 1:1000 solution should be diluted with ten parts of normal saline solution, the dose of the combined diluted material being: dogs, minims ii to viii (0.13 to 0.5cc); cattle and horses, ʒss to ʒii (2 to 8cc).

Adreno-Cortical Hormones

It has long been recognized that when animals are deprived of the medullary portion of their adrenal glands they may survive, but that if the entire adrenal tissue or cortical areas are removed the organism will die soon afterward, the survival period varying considerably with the species. Positive evidence of the life-sustaining activity of the adrenal cortex extracts was first obtained in 1928 by Rogoff and Stewart, who were able to extend the life-span of adrenalectomized dogs following administration of an aqueous-glycerin extractive called "interrenalin." By certain refinements in the extraction process, Swingle and Pfiffner obtained a cortical principle in 1931. Hartman and his associates, had earlier in 1928, suggested the name "cortin" for the hormone thought to be present, and this term is at present applied to the natural complex extractives which consist of a number of principles. As the result of chemical studies of Kendall, Reichstein, and Wintersteiner, over 20 crystalline derivatives have been isolated, and these complex compounds have been recognized as steroid derivatives closely related to hormones identified with the reproductive functions, e.g. progesterone. The most potent of the hormonal substances, first synthesized by Reichstein and later discovered actually to exist in the cortical extract, has recently (1937) been synthesized and called "desoxycorticosterone."[1]



Subcutaneous injections of epinephrine are sometimes used in the treatment of laminitis. Chronic laminitis usually requires surgical intervention

[1] Desoxycorticosterone is available in the form of the acetate under several trade names, e.g. doca, cortate, percorten. It is issued in the form of an oil solution in ampuls intended for intramuscular administration, each cc containing 5mg of desoxycorticosterone acetate. The oil solution is also available in the form of rubber-capped vials (10cc). Crystalline material is also available for the purpose of implantation subcutaneously. The formula is $C_{21}H_{32}O_4$.

That this substance does not represent the full activity of the whole complex, has been proved by experimental and clinical evidence. According to Grollman, a well-known authority on the adrenal functions, it is questionable if any of the adrenal-cortical hormones exert a single specific activity; in fact, they might be compared to vitamins in exerting manifold activities. For example, Kendall states that corticosterone, "compound E" and certain derivatives influence muscle efficiency and "gluconeogenesis" (glycogen formation); desoxycorticosterone appears to be essential for effecting electrolyte distribution; and an amorphous fraction maintains normal renal functions. Thus it is obvious that a multihormonal complex controls widely divergent functions.

Kendall outlines the physiologic activities of the adrenal cortex as follows: (1) Carbohydrate metabolism. (2) Capacity of muscle to respond to stimulation. (3) Distribution of electrolytes. (4) Renal function. (5) Growth of young animals. (6) Atrophy of adrenal and thymus glands (of normal rats). (7) Resistance to stress (bacterial toxins, low temperature, low oxygen pressure, etc.). Britton and Silvette in 1937 showed that following removal of the adrenal cortex, glycogenic stores in the liver and glucose in the blood serum reach very low levels. Long and others have demonstrated that in partially depancreatized rats either extracts of the cortex or corticosterone produces glycosuria. Long and others have also shown that the adrenal cortex increases the rate of gluconeogenesis, and certain factors in the extracts result in conversion of proteins to carbohydrate. In this respect, the various fractions show a wide divergence. Desoxycorticosterone is much less efficient in such conversions than certain of the other factors, hence does not improve carbohydrate metabolism in cortico-adrenal insufficiencies. Ingle, in 1936, devised a method of assaying cortical extracts by noting effects on muscular efficiency in adrenalectomized rats. Certain of the fractions favorably influence such activity, though again desoxycorticosterone has little effect. As regards maintenance of normal blood urea and normal condition in the bloodstream of adrenalectomized dogs, Kendall has found that desoxycorticosterone is six times more active than corticosterone. It has also been shown that desoxycorticosterone acetate, as well as desoxycorticosterone, is capable of increasing the concentration of sodium and decreasing the potassium concentration in the serum of normal rats more than any other factor in the cortex thus far isolated. Loeb has shown that potassium concentration can be suppressed to such low levels that paralysis will ensue. The portion of the cortical material which is so essential to life is known as cortin. The so-called amorphous fraction which remains after all crystalline material has been removed, has life-sustaining properties but has little influence on gluconeogenesis and muscular efficiency. However its influence on maintenance of the renal functions is marked. Only one or two gamma per kg of body weight is required daily to maintain adrenalectomized dogs.

When extracts of the adrenal cortex are fed to normal rats, it has been noticed that the adrenal as well as the thymus glands atrophy in a week or ten days, according to Ingle *et al.* The effect is specific only for certain factors, for it has been demonstrated by Wells and Kendall that adrenal atrophy produced by corticosterone and certain other compounds, is not produced by desoxycorticosterone; and little effect follows the use of the amorphous fraction. It has been shown that growth is retarded in adrenalectomized rats, but this effect again varies considerably with the factor in question. Resistance to stress has only recently been studied, and but little information is available regarding specific effects of the various factors on bacterial toxins, etc. It has been recently shown that the simultaneous administration of sodium chloride and desoxycorticosterone acetate affords the most protection against the effects of histamine. Certain factors in the cortical extracts protect adrenalectomized animals against the toxic influences of thyroxine. It has also been shown that corticosterone acetate prevents insulin convulsions. Selye and Schenker, confirming Hartman's work, have studied the influence of low temperature on adrenalectomized animals. The amorphous fraction, "compound E," and

corticosterone appear to be equally active in maintaining animals subjected to such conditions.

Thus it will be seen that the adrenal cortex extracts contain not one but a large number of closely related steroid compounds having specific effects which are qualitatively dissimilar. This has been proved by the fact that in adrenalectomized animals, true substitution can only be accomplished when each of the important disturbed functions is provided for by proper factors.

General Symptoms. — Following extirpation of both adrenal glands, one of the earliest and most striking symptoms is increasing asthenia. Immediately following the operation, the animal may show no ill effects, except perhaps inappetence; but later, and especially prior to death, the animal becomes depressed and incoordinate, shows weakness in moving about and is generally apathetic. There is pronounced fatigue on the least exertion, the extremities stiffen, and there is profound prostration, followed by dyspnea, cardiac feebleness, and fatal collapse. It has been observed that in rabbits convulsions are common, though this symptom is usually absent in the dog and cat. Rats resist the operative procedure for longer periods, probably because of the presence of accessory cortical bodies; hence chronic effects are more readily studied in these animals, which manifest impaired growth, poor fat deposition, delayed involution of the thymus, increase in weight of secondary sex organs, and at times degeneration of the renal tubules. Finally, the blood electrolyte balance is markedly disturbed, there being high values for urea and non-protein nitrogen, fall in blood sugar, increase in inorganic phosphate, decreased CO_2-capacity, fall in blood sodium and chloride, and rise in potassium and magnesium. Important to note is the fact that the fat, carbohydrate, and sodium-potassium-chloride balance is markedly disturbed. Water metabolism is also influenced.

Herrick and Torstveit have reported that destruction of the adrenal glands in male fowls results in marked reduction in size of the gonads and regression of the head furnishings. More recently, Herrick and Finerty have concluded that adrenalectomy in the fowl causes degeneration of the testes indirectly by way of the pituitary gland.

Standardization.—A number of methods of standardizing extracts of the adrenal cortex have been suggested but none has been accepted officially. One test, the one in which survival time of adrenalectomized rats is observed, is not specific for any one fraction. Another is the determination of growth or gain in weight in adrenalectomized rats, but this response is not very specific, though desoxycorticosterone shows gains beyond normal. The Everse-de Fremery test measures response of muscles to short stimulation; that is, duration and vigor of response are ascertained. Adrenalectomized rats either do not respond or react in a limited manner before muscular exhaustion occurs. In this test, desoxycorticosterone acetate produces about 10 times greater effects than corticosterone. A negative response, however, does not signify absence of cortical activity, since other criteria may show it. Ingle devised a prolonged muscular stimulation test, which is negative for desoxycorticosterone and positive for other fractions in the extract. This test is highly specific for certain cortical compounds. Pfiffner, Swingle and Vars have used the maintenance test in adrenalectomized dogs as an assay method. The unknown material is estimated as to its capacity to maintain the proper electrolyte balance of the blood, to maintain weight, appetite, etc. This is specific for the amorphous fraction and also for desoxycorticosterone, 15 gamma per kg of body weight being required to keep adrenalectomized dogs in normal condition.

Reviewing the end effects of adreno-cortical deficiency, it may be said that there is marked inability on the part of the kidney to function properly; water and sodium chloride are lost, while waste products such as potassium and urea accumulate in the blood stream. Liver function, as regards carbohydrate and cholesterol metabolism, is also impaired. Furthermore, the muscles are unable to contract normally and hence manifest marked asthenia.

In passing, it should be pointed out that overproduction of adreno-cortical secretion

is rare, but the effects are reflected in certain cortical neoplasms which have been observed to produce (in the human) masculinization—presumably due to production of androgenic substances. If the medullary portion is also involved, the tumor may give rise to paroxysmal hypertension due to hypersecretion of epinephrine.

In the rat, Selye and Dosne have found that overdosage with desoxycorticosterone acetate in normal animals (10mg daily) causes marked hypochloremia and adrenal atrophy. This is just the opposite of the effect in adrenalectomized animals. Kuhlmann *et al* have reported that dogs chronically treated with this substance in high dosage show effects the opposite of those encountered in adrenal insufficiency; namely, decrease in serum potassium, slight increase in serum sodium, decrease in serum protein and non-protein nitrogen. Ragan has noted that dogs develop diabetes insipidus, muscular weakness due to low potassium, and other effects following excess dosage.

Possible Applications. — The adrenal-cortex extracts such as cortin, or the synthetic substitutes such as desoxycorticosterone acetate, may have the following indications in treating lower animals (based on findings in the case of humans, including Addison's disease, a condition due to either tuberculosis or neoplasm of the adrenals). It should be noted, however, that since there are species variations which are sometimes surprising, the suggested indications are merely tentative.

1. Abnormal salt craving associated with adrenal insufficiency (shown by experimental animals which have been adrenalectomized as well as by humans with adreno-cortical deficiency).

2. Uremia. Selye has observed that desoxycorticosterone acetate used as pretreatment in experimental animals, prolongs the survival time of the animals in which acute uremia has been produced by bilateral nephrectomy. Incidentally, he also finds that the compound (due to its renotropic influence) protects experimental animals against the destructive action of corrosive sublimate (mercuric chloride).

3. Traumatic shock and allied conditions. Selye *et al.* have determined that following shock, there is a defense reaction which he terms "countershock"; and it is at this stage that shock symptoms are usually reversed. In adrenalectomized animals, this characteristic "countershock" reaction fails to develop. However, when cortin is administered to such animals the reaction to shock is normal and the organism becomes resistant to noxious agents. It appears that carbohydrate metabolism plays an important part in this complicated phenomenon; hence it is not surprising that cortical extracts are so effective. Desoxycorticosterone has proved to be of little value for this condition; reliance can be placed only on the active cortical extracts.

4. Asthenia. Hypoadrenia is often the cause of asthenia, and if this is the suspected condition, adreno-cortical extracts may be tried.

5. Dehydration. Since the degree of water retention is apparently related to the sodium content of the blood, which in turn is conditioned by adreno-cortical activity, it is logical to attempt the use of cortical extracts, or substitutive measures. In discussing disease of swine, Pinkerton has referred to the very recent work of Davidson, who employed adrenal cortex extract as a supportive measure in gastroenteritis and in other debilitating diseases where dehydration may occur. Horses and dogs suffer more frequently from dehydration than the other domestic animals but no cases in which adrenal cortex has been used in the treatment appear to have been reported.

6. Infectious states. Since these conditions are often followed by asthenia, cortin has been found useful (by Hartman). In chronic bacteremia, Neil found adrenal cortex helpful. In pyogenic infections, Wenner and Cone found such treatment beneficial.

7. Surgical procedures. Reed found better maintenance of blood pressure, body electrolytes, and cardiac functions when subjects were treated with adreno-cortical extracts pre- and post-operatively.

8. Acute toxemias, such as those due to burns and scalds.

Phenothiazine Poisoning

By C. D. FOLSE, Kansas City, Missouri

TOXIC effects of phenothiazine in horses, of sufficient seriousness to produce death, have been reported by Dr. T. T. Christian, Waco, Texas, Dr. John H. Woolsey, Hamilton, Missouri, and Dr. W. L. Hatcher, Cimarron, New Mexico.

Doctor Christian reports, that in the early part of November, 1940, he saw a pair of mares that had just come in from the races at Phoenix, Arizona. The history was one of intermittent appetite and diarrhea which began before they were shipped. Fecal examination revealed a heavy infestation with strongyles. Each of these mares was given two ounces of phenothiazine and a lesser dose was given to two younger animals in the string. All four responded well and by March, 1941, were back on the race track.

Because of the good results obtained, it was considered advisable to extend the treatment to seven other mares and three colts belonging to the same stable. Seven of the ten were medicated Jan. 2, 1941, and were off feed the next day. On the fourth day one was down and the other six seriously ill. The mare that was down died Jan. 7th, and another died four days later. A blood check on the 12th showed the percentage of red cells (determined by the hemocrit method) as low as 11.5% in one of the surviving horses—a Shetland mare. The red blood cell volume in the others was:

High Letty 13.4%.

Tret Black 21.0%.

Shetland colt 18.0%.

Since the normal volume percentage of erythrocytes is 33 to 35%, the percentage found in these animals evidences a severe and acute anemia.

Blood transfusions were made January 12th with phenomenal effects. Animals that had shown no disposition to eat for a week were reaching for food before the transfusion was complete. Normal horse serum used in one animal was effective, but the response was slow. One young animal recovered without treatment.

Investigation of all conditions that might be involved in the two sets of animals revealed a difference only in the feed. The first four animals treated in November, 1940, had received alfalfa, prairie hay, bran and oats. The others, which showed toxic effects, had been fed sorghum hay and omaline, which indicates the possibility that sorghum or sweet feeds do not go well with phenothiazine. Or perhaps animals require a ration well balanced as to protein to tolerate phenothiazine.

Doctor Woolsey reports that 23 horses weighing 1250 to 1800 pounds were given four ounces of phenothiazine powder per horse. It was given on feed containing alfalfa, molasses, cracked oats and coarse cracked corn. The roughage was soy bean hay with no beans, alternated with alfalfa, lespedeza, and timothy.

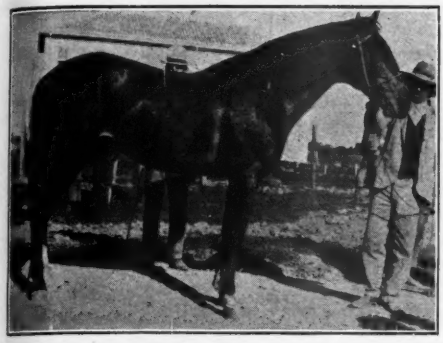
All of these animals were off feed the next day. They were gaunt, and began losing weight rapidly. In seven days the loss of weight was 200 to 250 pounds each. When they moved the gait was wobbly. There was complete loss of appetite and thirst, scant, ropy urine of the color of that of azoturia, and all mucous membranes were pale. Death claimed several of them within ten days. This case comes nearer to incriminating the molasses. There would have been no acute lack of protein in the ration of these animals.

In Doctor Hatcher's report[1] it will be remembered that the mixture fed these horses contained nearly 20% molasses. There we have molasses again. In all the fatalities so far reported (except from enormous experimental doses), molasses has been a component of the ration, and molasses was the only significant common component.

B. J. Errington of the Kentucky Agricultural Experiment Station states[2] that anemia of varying degree was produced in thoroughbred horses by doses of phenothiazine and that the anemia was greater upon the administration of a second dose

[1] Hatcher, W. L., 1941. Phenothiazine poisoning in horses. *No. Am. Vet.* 22:3, pp. 159-160.

[2] Errington, B. J., 1941. Phenothiazine as an equine anthelmintic. *Vet. Med.* 36:4, pp. 188-193.



within four weeks. He also reported albuminuria and hemoglobinuria following phenothiazine medication. His experiments with dosage indicate a dose much smaller than that previously recommended is effective. Others have confirmed this. There is considerable evidence that 40gm is as effective for a 1000-lb. horse as any larger quantity. This is but one-third the dose given where the fatalities resulted.

W. E. Swales of Canada states[3] that 50% of phenothiazine is oxidized in the alimentary tract of sheep, and suggests an active purgative at the time of medication, to distribute the drug quickly in order to prevent a large part of the oxidation. Experience has already suggested that phenothiazine be not given to animals already suffering from marked anemia. Perhaps until we learn more about it we should refrain also from administering it to animals that are receiving molasses or sugar in the diet.

The findings here summarized suggest that the effective therapeutic dose of phenothiazine is not yet definitely established, and that much smaller doses than now recommended may be effective if combined with a suitable purgative agent. It would also appear that certain factors yet unknown may influence toxicity and that the amount of sugar in the ration may be involved.

It appears that the anemia, even in serious cases, may be immediately corrected by blood transfusion.

[3] Swales, W. E., 1941. Phenothiazine and anthelmintic medication with special relation to parasitic diseases of sheep. Abst. in *Vet. Med.* 36:1, p. 28.

Puzzling Death of a Cow During an Intravenous Injection

The article "Epsom Salt Poisoning in Cows," by Dr. R. H. Smith of Lewisburg, Tenn., in the May 1941 issue (p. 273) reminds me of the following case:

Several years ago I was called to see a Jersey cow, six years old. The cow was down and unable to rise without help and staggered when moved about. Since she had calved several months previously, milk fever was not suspected, although the symptoms were similar.

Upon questioning the owner as to any home treatment he may have used, he replied: "I gave her some salts, about a pound and a half." Immediately Epsom salt poisoning came to my mind, for I had treated similar cases before. Not having any calcium gluconate with me, I proceeded to warm up a calcium chloride, sodium chloride, dextrose and magnesium chloride solution.

As I started to inject the chloride preparation, the cow being down, became very quiet. But, when I was half way through with the intravenous injection, she began to bawl loudly. In fact, it was a louder noise than I have ever before or since heard either a bull or a cow make. The injection was stopped immediately and the needle removed. The cow, after reaching an unusually high pitch in her bawl, gradually lowered the tone and was dead in about two minutes.

This outcome was embarrassing for me. I started telling the owner how unusual the action was from this treatment. I said: "You are sure you gave her only salts?" He said: "No, I gave her salt, not salts." Remember it was a pound and a half that he gave.

Well, the only explanation I could offer for the cow's reaction to the treatment was the over abundance of the chlorine radical both per os and intravenously, and no better explanation has occurred to me since. It seems too bad that a cow should be sacrificed for a misunderstanding of the "s" on the end of salt.

S. W. STILES.

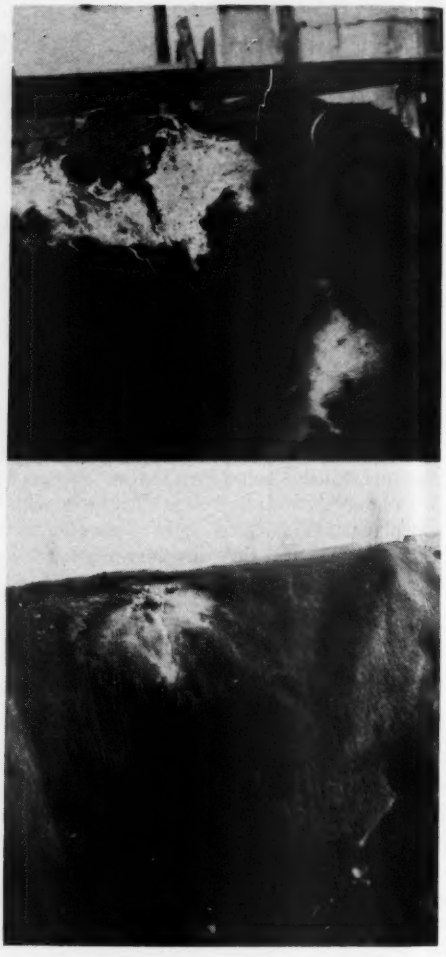
Falmouth Foreside, Me.

Injuries to Texas Cattle Caused By Starlings

If the hawk or the owl, recognized predators that feed upon mice, snakes, and sometimes poultry, should suddenly include in their prey the large farm animals, it would strike us as most strange. If in passing Farmer Brown's place we should hear him call to son Willie "Bring the shotgun quick—that old red-tailed hawk has caught another horse!" it would strike us as more than ludicrous; we would think that Farmer Brown probably needed the attentions of an alienist. What then shall we say of the bizarre action of the humble starling, heretofore content to glean after the herd, that suddenly begins to eat the cattle?

A report of injuries to cattle by starlings was made by Astle[1] in 1940. Under certain conditions, these birds are attracted to the wounds caused by the larvæ of the ox warble (*Hypoderma lineata*). Starlings are not flesh eaters ordinarily, but a taste of blood seems to incite them to further attacks on the wounds.

During late January and early February, 1939, a flock of five or six hundred starlings appeared around the feed lots and the pastures of a herd of dairy cattle near Dallas, Texas. Small flocks and individual starlings had been in this locality since late November. At this time the birds were observed picking up the seeds of Sudan and Johnson grass which scattered from hay fed to the cattle from the ground. Late in January a sharp fall in temperature with cold rains occurred, causing the surface of the ground to freeze. The birds, unable to obtain seed, started pecking at the ox warble larvæ protruding from the skin on the backs of the cattle. Apparently a taste of blood aroused them to further and more vigorous attacks on the wounds. Usually one bird began the attack upon an animal, but when blood began to ooze from the wound, often as many as 10 or 15 birds would flock to the carnage and in a few minutes enlarge the wound considerably. The resulting pain caused the animals to bellow, buck, and run under low-hanging limbs of trees in attempts to dislodge the birds. Often when the cattle were put in a barn for protection blood was draining from their injuries. After a few days the cold weather receded and most of the birds resumed their feeding upon the ground. On several occasions, however, the attacks were renewed. They had acquired a taste for raw meat!



Above: Injury about three weeks after attack. Note the sponge-like appearance of the granular exuberation. Below: Unsightly scar following healing of a lesion similar to that above

Lesions resulting from these attacks consist of large open wounds eight to ten inches in diameter. After about two days, swelling and suppuration occurred. Healing was slow

[1] Astle, Neville L., 1940. Starlings injure cattle. *Vet. Med.* 35:4, p. 235.

and after three or four weeks masses of exuberant granulations developed which in some cases protruded three or four inches above the level of the skin. Sloughing which left extensive open wounds followed. Prior to this sloughing of the flesh, which had a sponge-like appearance, the owner had not treated the wounds. However, as many flies appeared treatment became necessary. The application of commercial pine tar discouraged the flies but apparently prolonged the healing of the wounds. It was several months before the wounds finally healed, and large hairless scars were left upon the backs of the animals.

This was the first instance in this vicinity of such attacks by starlings. Perhaps the birds were migrating and were temporarily stranded at this ranch during the time the freezing temperature occurred. The incident reported here did not recur on the same ranch during 1941, nor have there been any other reports of injury caused by starlings to live stock in Texas.

JOHN W. McCOY, T.A.M C. '42
College Station, Texas

Tail Setting on the Way Out

The legislature of Massachusetts recently defeated an all-out attempt to repeal the law which makes it illegal to show horses with set up tails at fairs or public exhibitions within the commonwealth. New York State has a similar law; Michigan, a law prohibiting docking the tails of horses unless it be necessary. The A. V. M. A. took a stand a number of years ago against cropping the ears of dogs on the ground that it is a cruel, unnecessary practice. It is about time it considered the more cruel and equally unnecessary operation of tail setting. Recently a writer familiar with the subject stated in the *National Horseman* that only 10% of these operations of tail setting are performed by veterinarians and that the remaining 90% are performed by stablemen without anesthetics or any approach to asepsis. Obviously, the usual argument advanced by proponents of tail setting—that it is not unduly painful when properly performed by a veterinarian—does not apply to this 90%.

Leucoplakia In A Dog

During December, 1940, together with Dr. Horace Wilder of this city, I had occasion to examine a peculiar condition of the tongue

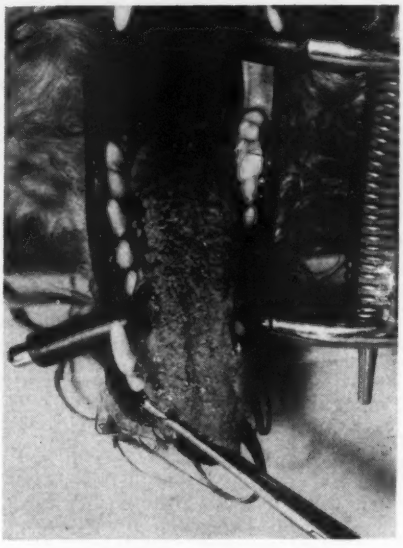


Fig. 1. The surface of the tongue was covered with pale, thickened areas

in a four-year-old, spayed female wire-haired terrier. The animal had shown symptoms of difficult deglutition and salivation for two years.

The surface of the tongue (Fig. 1) was covered with many pale, thickened areas, and the edges were swollen and red. Neither the throat nor the oral mucosa was affected. The animal was destroyed and sections of the tongue were examined microscopically.

The epidermis on the dorsal surface was thickened and, in areas, hyperkeratosis was evident. The lower layers of the Malpighian stratum contained foci of monocytes and lymphocytes, some of which filled the dermal papillae. Numerous embryonic hair follicles were noted in the corium and in tangential sections through the epidermis. Polymorphs were clustered around these foreign structures.

Foreign body reaction with resulting glossitis and leucoplakia is the suggested diagnosis.

ROBERT B. McCLELLAND
Buffalo, N. Y.

Canine Tetanus

On the afternoon of December 21, 1940, a three-year-old female Boston terrier was presented at the hospital of Drs. Collins & Collins at West Point, Nebraska.

The owner stated the dog had been sick for several days, the main symptoms he had observed being a peculiar stiffness and loss of appetite. On examination we noted an increased reflex excitability; the animal was very alert and seemed continually poised for action. Pulse, respirations and temperature were normal; the mucous membranes appeared healthy. A study of the habitus revealed extension of the limbs with the head drawn backwards (opisthotonos) due to tonic convulsion. The dog's forced walk somewhat resembled the German goose step. The facial expression was distorted (risus sardonicus) with the mouth commissures drawn backward and the forehead wrinkled. The eyeballs were slightly protruded and the nictitating membranes prolapsed. The bitch was grinding her teeth.

The owner said that the dog had been in a fight with a sow several weeks previously and had several "small scratches" on her, but examination failed to reveal any open wound lesions. The continuous character of the convulsions and the slow onset of the condition served to differentiate it from strychnine poisoning. In eclampsia, reflex excitability is not increased, and in any case the bitch was not pregnant—in fact, had never had puppies. The logical conclusion was diagnosis of tetanus.

The initial treatment consisted of injection of nembutal intravenously to relax the body musculature, and 10,000 units of tetanus antitoxin subcutaneously. The dog was placed in a cage in a quiet spot. The following day 5,000 units of tetanus antitoxin was given subcutaneously, and glucose and physiological saline solution intravenously. The third day, the dog received 5,000 units of tetanus antitoxin subcutaneously and was fed a small quantity of milk and ground beef, the food being placed on a box to enable her to gain access to it. Some relaxation of the rear parts was noted. The fourth day, relaxation was still more marked, the dog's appetite was better, and treatment was discontinued. The sixth day, the dog was still stiff, but her general condition warranted discharge from the hospital. When the owner brought the dog in for a checkup two weeks later, she had lost all signs of her encounter with *Clostridium tetani* and its potent toxins.

C. C. COLLINS.

West Point, Nebraska

* * * *

Sulfapyridine in the Treatment of Feline Distemper

Concurrently with observations on the use of sulfapyridine in the treatment of canine distemper and its complications,[1] a few observations were made on its use in cats suffering from feline distemper. This disease is apparently of virus origin, and complications, particularly of the respiratory tract, frequently develop and lead to a fatal termination.

1. Tabby cat, Sept. 15-17, 1940. Age 6 months. Feline distemper; temperature 104.2°F. Treated with 10gr of sulfapyridine a day; recovered in 3 days, with no untoward sequelae.

2. White cat, Sept. 6-10. Bronchial distemper; temperature 103.5°F. Twelve grains of sulfapyridine was administered daily, and recovery was complete in 4 days.

3. Siamese cat, Aug. 26-29. Age 4 months. Mild catarrhal distemper; temperature 103.2°F. Sulfapyridine was administered at the rate of 15gr a day. The cat developed a violent reaction to the drug, with face and pendant parts of skin edematous and eyelids swollen nearly shut. The dosage was reduced one-half; the swelling subsided and the cat recovered in 3 days. The animal had two slight convulsions during treatment.

4. Alley cat, Aug. 8-11. Age 6 months. Catarrhal distemper; temperature 103.8°F. Uneventful recovery followed sulfapyridine therapy.

We have also used sulfapyridine in five recent cases of feline distemper, with equally good results. Since this disease usually runs a protracted course and frequently terminates fatally, the sulfapyridine therapy appears distinctly promising. The drug was administered *per os* in 2½ to 4gr doses, three to six times a day. No toxic reactions were observed except in No. 3.

ARTHUR H. BRYAN.

Baltimore, Md.

[1] Bryan, Arthur H., 1941. Clinical observations on the use of sulfapyridine in the treatment of canine distemper. *Vet. Med.* 36:7, pp. 365-367.

Abstracts

Length of Gestation and Birth Weight in Cattle

A study[100] was made of the influence of the following factors on gestation period in cows and on birth weight of their calves: Sex of calf, individual differences in cows, breed, calving sequence, weight of dam, and season of calving. This study, carried on from 1932 to 1938 at Beltsville, Md., included 164 parturitions from 65 beef Shorthorn cows and 133 parturitions from 68 Milking Shorthorn cows. The average gestation period for the beef Shorthorn cows was 280.8 days and for the Milking Shorthorn 281.7 days. The respective mean birth weights of their calves were 69.2 and 79.2 pounds.

A probably significant difference between sexes in length of gestation was observed in the beef shorthorn cows but not in the milking shorthorns. Differences in length of gestation period account for 25 to 39 percent of the variation in birth weight between sexes.

There is a tendency for individual cows to have a characteristic length of gestation period, and the birth weights of calves produced by any one cow tend to be less variable than those of calves from different cows. Birth weight was found to be influenced by length of gestation, and cows which tend to carry calves overtime also tend to have heavier calves.

Breed appeared to have no effect on length of gestation, but in birth weight of calves there was a highly significant difference between breeds.

A multiple correlation indicates that the combined influence of length of gestation, calving sequence, and weight of dam account for 38% of the variation in birth weights in cattle. Length of gestation had the greatest effect of the three factors.

Season of calving had little influence on length of gestation or on birth weights.

[100] Knapp jr., Bradford, W. V. Lambert and W. H. Black, 1940. Factors influencing length of gestation and birth weight in cattle, *Jnl. Agr. Res.* 61:4, pp. 277-286.

Chemotherapy of Strangles

Sulfanilamide shows a positive protective effect in mice against experimental infection with the strangles organism (*Str. equi*).[103] Provided treatment is begun during the acute septicemic phase of the infection, rapid recovery of horses from strangles may be obtained by giving, per os, daily for at least six successive days, 0.1gm of sulfanilamide per kilogram of body weight. It is much less effective if used late in an attack, particularly if important lesions (especially pulmonary abscesses) are already in evidence.

* * * *

A Probable Agent for the Transmission of Fowl Paralysis

Brown and Cross of the Texas College of Arts and Industries report[106] that fowl (range) paralysis first appeared at that institution in 1937, when 61 of a flock of 225 chickens died of it. The following year there were 92 fatalities in a flock of 345; and in 1939, 403 of a flock of 439 birds were lost in a period of 4½ months.

In the latter year the fowls had access to an old barn which was heavily infested with "blue bugs," *Argas persicus (miniatus)*, and it was noted that practically all the birds were infested with these ticks. A number of experiments were then undertaken to determine the responsibility of the ticks for communicating the disease from sick to well birds. In experiments where sick and healthy birds were associated, the disease spread rapidly if ticks were present, but the healthy did not become infected if raised in disinfected pens in which no ticks were present. When healthy birds were kept in pens infested with ticks they developed paralysis even though no paralyzed birds were introduced into the pens. Suspensions of ground ticks taken from a paralyzed bird produced paralysis when injected into healthy birds protected from contact with either sick birds or live ticks.

[103] Descazeaux, J., R. Courtade, and Rocq, 1939. La para-aminophénylsulfamide (1162 F) dans la chimiotherapie des infections par le streptocoque gourmeux. *Bull. Acad. Vet.* 12:7, p. 307; abst. in *Rev. de Path. Comp.* 40:518.

[106] Brown, J. C., and J. C. Cross, 1941. A probable agent for the transmission of fowl paralysis. *Science* 93:2422, p. 528.

Book Reviews

Animal Pathology. *By Russell A. Runnells,* D. V. M., M.S., Associate Professor of Veterinary Pathology, Iowa State College. Cloth bound; 604 pages, 184 illustrations. The Iowa State College Press, Ames, Ia., 1941. $6.00.

The first edition of this welcome text was reviewed[1] by the late Dr. L. Enos Day. At that time the chief criticism of the work was its brevity. That has been in considerable part remedied in the present edition, in which the text has been extended to 124 additional pages, or more than 25%, and at no increase in price. Specifically, the more important changes made in the second edition include:

The number of illustrations has been increased from 127 to 184.

Chapter 1 has been enlarged considerably in order to give the student a more complete conception of the scope of animal pathology.

Major portions of Part I have been rewritten and enlarged to include improvements suggested by Dr. E. A. Benbrook, Iowa State College.

The chapter dealing with food deficiencies has undergone the most extensive revision.

An introductory section has been added to each chapter in Systemic Pathology. In it the various functions of the system under discussion are briefly reviewed, and ways in which these functions can be disturbed by pathologic processes are pointed out.

An introduction of a general nature dealing with the nomenclature, etiology, transmissibility, channels of infection, period of incubation, pathogenesis, lesions, course and termination of the specific infections has been added to the discussion of the specific infectious diseases.

An appendix outlining a desirable technic for post-mortem examination and describing simple laboratory procedures for the laboratory tests most commonly used has been added.

This work is intended principally for students, but it will prove useful to practitioners also in enabling them to keep abreast of progress in matters pathological.

[1] *Vet. Med.* 34:6 (June, 1939), p. 392.

Eggs and Egg Products, a circular compiled by the Poultry Committee of the U.S.D.A., is one of 38 new publications issued recently by the U. S. Department of Agriculture. Although the circular may be of general interest to poultry and egg producers, it is written in semi-technical style and primarily for those who handle and work with eggs after they leave the producer's hand. Among the items covered are: nutritive value of eggs and their use in cooking, eggs in relation to Federal food and drug laws, the use of eggs and chick embryos as cultural media, uses of unedible eggs, the influence of breeding and feeding on egg quality, oil treatment of shell eggs, dried and frozen egg production, chemical composition of eggs, eggs in the manufacture of food products, cold storage and grading of eggs, and care of eggs on the farm. The circular (C 583) may be obtained from the Superintendent of Documents, Washington, D. C.; price, 15c.

* * * *

Colorado's Poisonous and Injurious Plants is the subject of a recent bulletin (No. 455) by Durrell and Newsom of the state experiment station. More than 30 years ago Glover of the same station wrote a bulletin, perhaps the earliest on plants poisonous to live stock in which he described 30 range plants that had been responsible for injury to cattle and sheep in Colorado. The present bulletin contains the illustrations of 95 such plants and describes without illustrations 17 others. In all 112 Colorado plants are thus known to be or are strongly suspected of being poisonous or otherwise injurious to live stock.

The discussion is clear, concise, pleasing and forceful. The botanical and common names of the plants are given with a brief description, the season at which they are injurious, the localities in which they grow, the circumstances under which they are usually eaten by live stock, the symptoms, the treatment, means of preventing injury by the plants and, where practical, of eradicating them. In addition, excellent drawings are included of 90% of the plants described.

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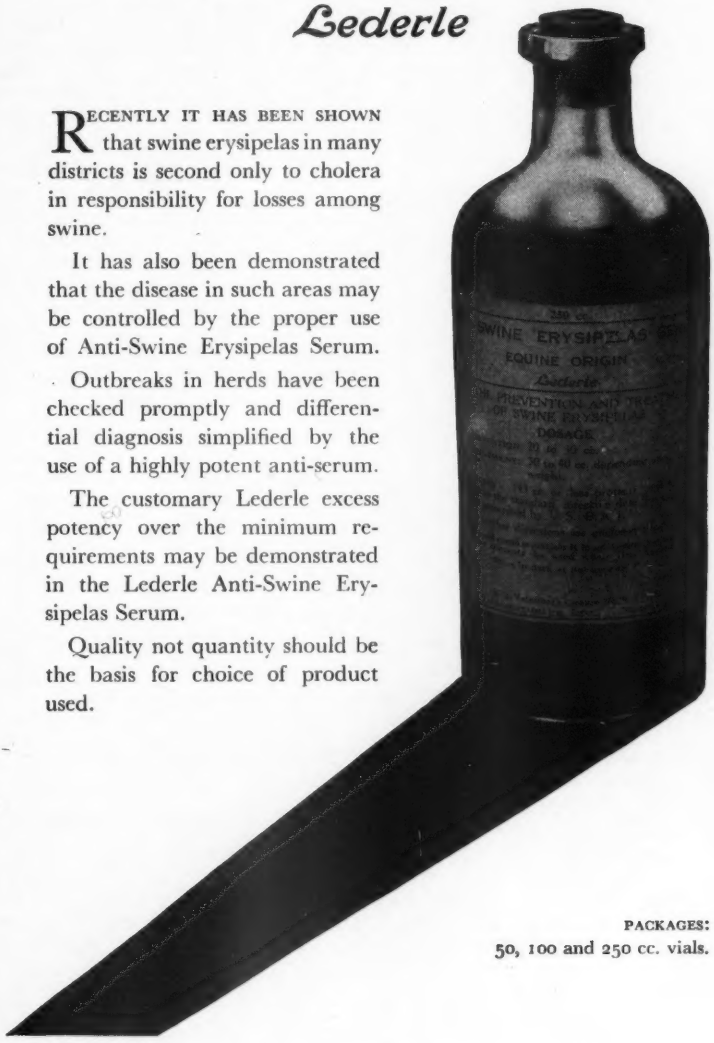

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■

Carter H. Anthony has resigned from the Department of Bacteriology and Veterinary Science at the University of Arkansas to accept a Civil Service appointment as veterinarian at the National Zoological Park, Washington, D. C., under Dr. W. M. Mann, Director.

■

Roy E. Nichols

Roy E. Nichols, since 1933 instructor in the department of surgery and clinics of the College of Veterinary Medicine, Ohio State University, has joined the Purdue University Veterinary Department as Veterinary Physiologist. Doctor Nichols was graduated from Ontario Veterinary College in 1933, received a master's degree from Ohio State in 1934 and his Ph.D. from the same institution this year.

■

After 11 years with the Old Trusty Dog Food Company, W. E. MacQuarrie has resigned as General Sales Manager to join the Gaines Food Company. Working out of Boston, Mr. MacQuarrie will cover eastern Massachusetts, Rhode Island, Hartford County in Connecticut and the three southern counties of New Hampshire. Another recent addition to the Gaines sales organization is Jack Quinlan, who has been assigned to the Minneapolis territory. Mr. Quinlan was previously associated with Archer-Daniels-Midland, as general sales manager of the Airy-Fairy Foods Division.

Last month we mentioned two veterinarians prominent in the milk industry. . . . Another is Dr. Robert Prior of Seattle, Washington, Manager of the Washington State Dairy Products Commission and Vice-President of the American Dairy Association.

■

Science sometimes goes to extraordinary lengths in its quest for knowledge. . . . Tests made recently on Wah, the 4,000-year-old Egyptian mummy of the American Museum of Natural History, prove beyond the shadow of a doubt that the venerable old gentleman had type B blood.

■

According to Prof. Julian Huxley, curator of the London Zoo, most species of birds have grown used to the air raids on England. The reactions of parrots have varied. One is said to have learned to imitate the sirens, another fell into a deep mood of melancholy, and a third was so upset that it lost its power of speech for weeks.

■

Closer supervision over the distribution of viruses, serums, and similar biological products manufactured under federal licenses will be required by the U. S. Department of Agriculture after January 1, 1942, under Amendment 15 to B. A. I. Order 276. After January 1st distributors, as well as producers and importers, will be required to keep permanent records concerning any veterinary biological products they handle, and to forward notices of all shipments of virus, serum, toxin, etc., consisting in whole or in part of live virus or live organisms, to the federal B. A. I. and to the proper live stock sanitary official in the state to which the shipment is consigned.

■

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Coal, air and water are used to produce ammonia, which is used in making ice; the same three products are utilized in making antifreeze. Chemically combined, these commonplace substances are also the materials used in the manufacture of nylon, which is replacing silk for many purposes. (Incidentally, the invention of nylon brought Dr. C. M. A. Stine the Pekin Medal of the American Section of the Society of Chemical Industry.) . . . It seems that there is no limit to the possibilities, in the hands of modern scientists, for the creation of new and even more miraculous uses for combinations of coal, air and water.

Coming Meetings

Kansas City Veterinary Medical Association, Kansas City, Mo., July 21.

San Diego County Veterinary Medical Association, Zoological Research Building, Balboa Park, San Diego, Calif., July 21.

District of Columbia Veterinary Medical Association, Mayflower Hotel, Washington, D. C., July 22.

Iowa Veterinary Medical Association, second annual golf and stag day, Pine Lake Country Club, Eldorado, Iowa, July 31.

American Veterinary Medical Association, Murat Theater and Shrine Temple, Indianapolis, Ind., August 11-15.

Eastern Iowa Veterinary Association, 28th annual meeting, Hotel Montrose, Cedar Rapids, Iowa, October 7-8.

University of Illinois 22nd annual veterinary conference, new Union Building, University of Illinois, Urbana, Ill., October 14-15.

Purdue University Short Course for Veterinarians, Lafayette, Ind., October 15-17.

First of its kind is a market hog and pork products show sponsored by the National Swine Growers Association. It will be held with the Illinois State Fair in Springfield, August 16-24, 1941.

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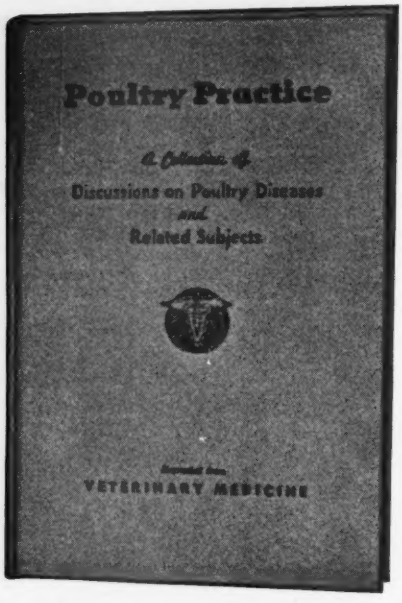

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Army Veterinary Corps

Transfers.—Lt. Col. Seth C. Dildine, from Fort Lewis, Wash., to Chicago Quartermaster Depot, Chicago, Ill., July 15; Lt. Col. Solon B. Renshaw, from Fort George G. Wright, Wash., to Fort Lewis, Wash., July 10; Lt. Col. Raymond I. Lovell, from Presidio of Monterey, Calif., to Camp Forrest, Tenn., July 20; Maj. Ralph W. Mohri, from Panama Canal Department to Second Cavalry Division, Fort Riley, Kans.; Maj. Arvo T. Thompson, from March Field, Calif., to Headquarters Fourth Air Force, Riverside, Calif.; Maj. Ernest E. Hodgson, from Headquarters Southwestern Remount Area, Colorado Springs, Colo., to Headquarters Third Air Force, Tampa, Fla., July 15; prior to proceeding to Tampa, to the Chicago Quartermaster Depot for temporary duty of approximately one month (course of instruction); Capt. Wm. E. Jennings, from Front Royal Quartermaster Depot, Front Royal, Va., to Fort Sill, Okla., Aug. 26; Capt. James B. Nichols, from San Francisco Port of Embarkation, Fort Mason, Calif., to Headquarters Second Air Force, Fort George Wright, Wash., July 10; Capt. Richard G. Yule, from Fort Leavenworth, Kans., to Headquarters First Air Force, Mitchell Field, N. Y., July 10; Wayne D. Shipley, from University of Kentucky, Lexington, Ky., and additional duty at Headquarters East Central

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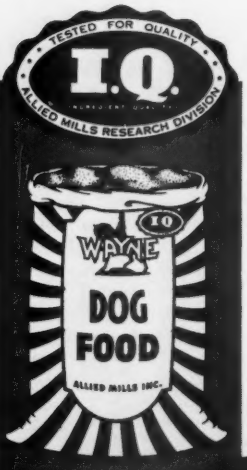

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Promotions.—To major: Capt. Harvie R. Ellis (major, Army of the United States) June 24.

Temporary Promotions.—To colonel: Lt. Cols. Mott Ramsey, Gerald W. Fitzgerald, Allen C. Wight, Elwood L. Nye, Chauncey E. Cook, June 26; to major: Capt. Robert P. Kunnecke, retired, May 23.

Veterinary Corps Reserve

Transfers.—Maj. John W. Burke, from Medical Field Service School, Carlisle Barracks, Pa., to Chicago Quartermaster Depot, Chicago, Ill., July 15; Capt. Arthur C. Zedlitz, from Fort Sam Houston, Tex., to the Fort Worth branch of the San Antonio General Depot, Fort Worth, Tex., July 10; Capt. Richard E. Geisler, from Fort Douglas, Utah, to Headquarters Northwestern Remount Area, Salt Lake City, Utah, Aug. 1; Capt. Arthur C. Jerstad, from Fort George G. Meade, Md., and Herbert F. Sibert, from Fort Sill, Okla., to Kansas City Quartermaster Depot, Kansas City, Mo., June 30; 1st Lts. Norman H. Stoner, from Fort Brady, Mich., to Selfridge Field, Mich.; Mertin L. Tuttle, from Fort Hayes,

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Ohio, to Fort Benning, Ga., June 25; Karl F. Steinbach, from Army Medical Center, Washington, D. C., to Sixth Corps Area Laboratory, Fort Sheridan, Ill., July 15; Edward H. Von Glan, from Fort Lewis, Wash., to Hawaiian Department; Benners B. Vail, Jr., from Camp Shelby, Miss., to Panama Canal Department, leaving New York July 8; Ervin A. Eichhorn, from Army Medical Center, Washington, D. C., to Headquarters First Corps Area, Boston, Mass., for duty with the First Corps Area Laboratory, July 25; James I. Cornwell, from Fort Bragg, N. C., and Lester J. Gorman, from Fort Mayer, Va., to Kansas City Quartermaster Depot, Kansas City, Mo.

Revocation of Order.—Special order relieving Capt. Earl T. Martin from assignment and duty at Fort Ord, Calif., and assigning him to duty in the Hawaiian Department, is revoked.

Promotion.—To captain: 1st Lt. Edwin M. Crawford, June 18.

Ordered to Extended Active Duty.—Maj. Colenzo H. Hoffmire, to First Armored Division, Fort Knox, Ky.; 1st Lts. Philip A. Lee, to Mather Field, Calif., and Willard M. Van Sant, to Air Corps Gunnery School, Boise, Idaho.

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Meat Inspection Course.—The following officers, now on active duty, are ordered to report at the Chicago Quartermaster Depot June 9, 1941, for a 30-day course of instruction in the inspection of meat, meat-food, and dairy products: Maj. Philip H. Fulstow, Pine Camp, N. Y.; 1st Lts. James H. Gillespie, Air Base, Bangor, Me.; Joseph J. Merenda, Fort Slocum, N. Y.; Charles B. Frank, Fort Myer, Va.; William A. Nusser, Fort Story, Vo.; Aaron R. Griffith, Camp Davis, N. C.; Russell S. Wann, Camp Croft, S. C.; Bernard F. Mood, Camp Wheeler, Ga.; Philip C. Manly, Fort Knox, Ky.; Russell M. Madison, Chanute Field, Ill.; Ross H. Hurt, Camp Grant, Ill.; Dale R. Stephenson, Fort Custer, Mich.; John M. Dermody, Fort Francis E. Warren, Wyo.; Henry Loughridge, Fort Meade, S. D.; Charles R. Wainright, Brooks Field, Tex.; Charles E. Robinson, Lowry Field, Colo.; William B. Snodgrass, Fort Ord, Calif.; John L. McKitrick, Fort Hayes, Ohio; George A. Leonard, Hamilton Field, Calif.

National Guard

Maj. William A. Shannon, Camp Edwards, Mass., is ordered to report at the Chicago Quartermaster Depot June 9, 1941, for a 30-day course of instruction in the inspection of meat, meat-food, and dairy products.

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The Kansas Horse Plague and Encephalomyelitis .....J. R. Zecha
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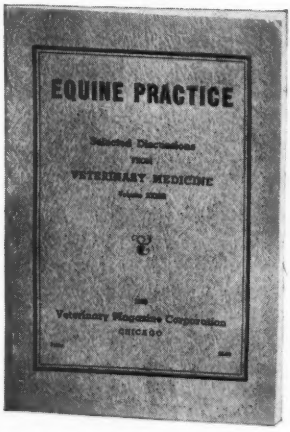

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Deaths of Veterinarians

C. M. Riley, Portland, Ore., McKillip Veterinary College '09, B.A.I., February 16.

G. A. H. Edmiston, 62, Easton, Md., McKillip Veterinary College '14, March 23.

Erman C. Williamson, 49, Montpelier, Ind., Indiana Veterinary College '15, April 1.

S. S. Gibson, Randolph, Nebr., McKillip Veterinary College '17, April 14.

W. E. A. Wyman, St. Petersburg, Fla., New York State Veterinary College '95 and McKillip Veterinary College '98, April 18.

William J. Tower, 23, Leominster, Mass., Ontario Veterinary College '39, April, 1941.

Victor Herbert Harris, Ruston, La., McKillip Veterinary College '16, May 3.

Harry C. Stamen, 44, New Rochelle, N. Y., Ohio State University '22, on the staff of the Angell Memorial Hospital, Boston, for five years before entering private practice at New Rochelle; served for several years as a director of the New Rochelle Humane Society, May 4.

P. E. Nulph, 64, Campbellsport, Wis., Grand Rapids Veterinary College '12, practiced in the Dakotas and Minnesota until 1923, when he came to Wisconsin; in B.A.I. service for the last seven years, May 5.

Ralph R. Shaw, 47, St. Paris, Ohio, McKillip Veterinary College '16, May 6.

C. W. Rippon, 49, Heart Lake, Pa., George Washington University, World War veteran, former chief field veterinarian for the Pennsylvania B.A.I., May 11.

William C. McLean, 60, San Mateo, Calif., San Francisco Veterinary College '01, May 18.

J. H. A. Anderson, Monticello, Ind., Terre Haute Veterinary College '13, May 29.

R. F. Rasmussen, Montevideo, Minn., Ontario Veterinary College '33, May 30.

James E. Robertson, 88, Seattle, Wash., Ontario Veterinary College '94, May 30.

A. E. Robertson, 68, Nashville, Tenn., former city meat inspector, June 2.

Clyde Everett Warden, 45, Mount Hope, W. Va., world war veteran, June 5.

Charles R. Bracke, 51, Belleville, Ill., June 8.

David E. Hickman, 59, West Chester, Pa., University of Pennsylvania '04 June 10.

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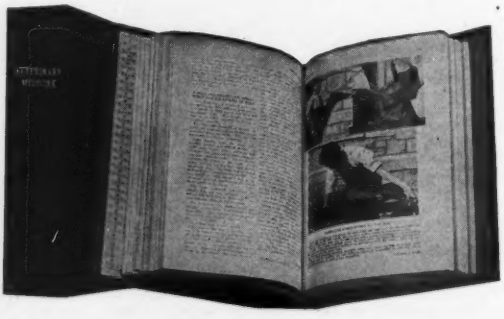

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J. Franklin Stevens, 81, West Liberty, O., June 14.

John A. O'Connell, 65, Dorchester, Mass., Harvard Veterinary College '96, world war veteran, chief veterinarian of the Boston Health Department, June 19.

Hubert C. Carver, Higginsville, Mo., Kansas City Veterinary College '11, practiced in Higginsville for 30 years, June 24. His son, Hubert E. Carver, Texas A. & M. College '41, is taking over his practice.

H. G. Atwood, 69, president of Allied Mills, Inc., died June 27 in Rochester, Minn., following an operation. Mr. Atwood, who made his home in Peoria, Ill., had been in the grain business for more than 40 years.

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